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THYROGLOSSAL CYSTS AND FISTULAE*

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THE gifts of embryology to surgery are manifold. The thyroglossal tract, representing not the least of these, was certainly one of the first to be generally appreciated. For this paper it was considered advisable to adhere strictly to the surgical aspect of thyroglossal cysts and fistulae.

In hospital surgical practice thyroglossal cysts are frequently encountered. The actual incidence in the general run is probably exceedingly small, for, according to Sistrunk, in eighty-six thousand consecutive patients examined in the Mayo Clinic, only thirty-one cases of thyroglossal cyst were found. In order to understand thoroughly the diagnosis and treatment of this condition one must fully understand the embryology of its development.

EMBRYOLOGICAL ANATOMY

The divergent views of the embryologists concerning the development of the thyroid gland and the tongue have left many questions undetermined. It is now generally believed that the thyroid gland arises from a single, median primordium (or anlage), and that the tuberculum impar is not the parent structure of the anterior tongue segment (as was taught by His), but that this is formed from two median projections near the anterior ends of the mandibular arch. The foramen caecum marks the situation of the starting point of the thyroid primordium. It may be the remnant of the primordium, as is commonly believed, or it may represent the final point of fusion of the double anterior and posterior segments of the completed tongue. The thyroid gland primordium begins in the floor of the pharynx at the tuberculum impar, early in the fourth week of embryonic life. It begins as a short canal-like diverticulum, which fuses to form an epithelial cylinder without a lumen, and this structure separates from the pharynx and grows downward into the neck, anterior to the region of the hyoid bone, whose development occurs later. The thyroid gland develops in the depth, but the remainder of the thyroglossal tract regresses.

The hyoid bone begins to develop at about the beginning of the fifth week, and lies in close apposition with the thyroglossal tract. As the hyoid bone grows forward, the inelastic thyroid tissue is compressed and obliterated at the point of contact, and the tract is divided into suprahyoid and infrahyoid segments. A later continued hyoid growth, forward and slightly downward, produces a convexity in the bone in the sagittal plane, and by this growth the upper end of the lower segment of the thyroglossal tract is again divided, a small portion tending to remain on the anterior hyoid surface, the next small segment disappearing under the pressure of the lower bone ridge, and the top of the lowest segment being carried far back almost behind the bone, owing to its close attachment to the lower border of the hyoid, which now curves backward. In this manner the foundation is laid for the appearance in postnatal life of thyroid tissue (1) in the region between the root of the tongue and the hyoid bone, (2) upon the hyoid bone and (3) below and almost behind the hyoid bone, in closest relation to it, as well as lower in the midline of the neck.

Coincidently with this development of the thyroglossal tract and as a consequence of its rapid growth, small segments of the normal lining membrane of the pharynx about the primordium of the thyroid gland are torn loose and carried for varying distances into the depth. These segments have all the tissue elements of the mucosa and of these the important structures are the epithelium, the glandular elements, and lymphoid tissue. The epithelium may be squamous or ciliated. This tissue persists in the fetus in the form of small cysts which lie in the path of the thyroglossal tract. They are numerous in the region of the root of the tongue and diminish rapidly in number toward the depth. The complex relation of these cysts with the hyoid bone has been a matter of careful examination. The thyroglossal tract and the hyoid bone are developed before the muscles and fascia attached to the bone appear. The thyroid tissue and the associated tissue elements from

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the floor of the pharynx are thus in very intimate connection with the bone. As the muscles and fasciae appear and competitive growth begins, the weaker thyroid epithelium tends to atrophy, but the more vigorous mucosal epithelium advances, and may also grow into the periosteum and may erode the hyoid substance—but this process, even though extensive, occurs from the anterior surface of the bone, and there is no "perforation" of the hyoid by the tissue of the thyroglossal tract.

The early belief of His that there was a patent lumen in the cylinder of thyroid epithelium led him to give it the name "thyroglossal duct," and this erroneous teaching has been continued in textbooks to the present time, despite its correction by His, in 1891, and other conclusive studies, notably those of Streckeisen and of Wenglowski. At the present time it may be stated that the postnatal pathologic processes that arise in the vestigial remnants of the thyroglossal tract are of two types,—the thyroid tissue producing median thyroid adenomata alone, and the related mucous membrane fragments producing median cysts and median fistulae with or without adenomatous or lymphoid additions.

CLINICAL CONSIDERATIONS

The pathologic conditions found in postnatal life that seem to have their origin in vestigial rests of the thyroglossal tract and its inclusions, are:

(1) Heterotopic masses of thyroid tissue in the root of the tongue (lingual goiter), or above or below, and rarely in front of the hyoid bone.

(2) Median cysts in the tongue, under the tongue, and above, about, or below the hyoid bone.

(3) Median fistulae.

These may be single or they may occur in combination.

HETEROTOPIC THYROID ADENOMATA

These may occur at any point in the thyroglossal tract, but are seldom found above the hyoid bone. They seem to have no clinical importance beyond the presence of the small tumor, presumably because the fetal character is largely retained and differentiation has not progressed very far. When there are two pyramidal tracts, the tumor will lie at the corresponding side of the midline. It is of importance, before removing these heterotopic thyroid masses, to determine that other thyroid tissue is present in the normal site, since the heterotopic mass may be the only thyroid tissue present. The diagnosis is fairly obvious and excision is simple, since there is no firm fixation.

MEDIAN CYSTS AND FISTULAE

These constitute a more important problem than the heterotopic thyroid tumors because of their greater frequency, more complicated structure and relations, and their tendency to recur after operation. The characteristics found in the cysts of postnatal life are very similar to those of the embryonic period, but they both are very different from those relating to the branchiogenetic cysts and fistulae. They are derived wholly from epithelial inclusions torn away from the lining of the floor of the mouth by the rapid growth of the thyroid primordium, but the thyroid tissue has no other relation to these cysts and fistulae, and it is never found as a part of the wall or the lining membrane of the cysts or fistulae. Thyroid tissue, however, may be found in the neighborhood in some cases. No complete thyroglossal fistula with outer skin orifice and inner pharyngeal opening has ever been reported.

Median cysts and fistulae have not been found at birth but develop later, mainly in childhood and adolescence, but some also in adult life; few, however, after the age of thirty years. The cyst is the type structure and this becomes a fistula only after an acute inflammatory process or a surgical incision has produced the opening. Its onset may be gradual, a slowly growing cyst being the only manifestation, or there may be an acute inflammatory process without evident cause, which leaves the persisting fistula after its subsidence. Once developed, the fistula will not close or remain closed until the entire epithelial lining has been removed.

DIAGNOSIS

The diagnosis of thyroglossal cyst is usually simple. Certain difficulties are met with from time to time, however, the chief of these resulting from a peculiar liability of these cysts to infection. If a thyroglossal cyst comes under observation for the first time when thus complicated, its true nature is easily overlooked and a diagnosis of some inflammatory condition is made, notably, an abscess connected with the pharynx or paratracheal gland. Thus frequently when fluctuation cannot be obtained because the cyst is tense and small, an enlarged gland enters the question. Again, in the median line, a simple epidermoid cyst necessitates a difficult but an all important differential diagnosis. In this connection it should be mentioned that thyroglossal cysts frequently move upward when the tongue is fully protruded, and this may serve to distinguish them from epidermoid cysts. The translucency test is worth trying, but as only about 5% of the thyroglossal cysts are translucent, no importance can be attached to a negative result. Lastly, a diagnosis of thyroglossal cyst is never complete without definitely determining the presence of a thyroid gland in its normal position, for an ectopic thyroid has many times unwittingly been removed. Not infrequently this thyroid is the only thyroid for

that individual. Remnants of thyroid tissue along the course of the thyroglossal tract have an inverse relationship to the normal development of the normal thyroid gland.

LOCATION

Thyroglossal cysts and fistulae may occur anywhere along the course of the thyroglossal tract, that is, from the foramen caecum to the body of the thyroid gland. For purposes of description, these locations have been tabulated as follows:

1. Beneath the foramen caecum.
2. In the floor of the mouth protruding beneath the chin.
3. Suprahyoid.
4. Subhyoid.
5. On the thyroid cartilage or membrane.
6. At the level of the coracoid body in the suprasternal notch.

The first three of these situations have been found extremely rare. The great rarity of thyroglossal cysts and fistulae in these locations must make one scrutinize very closely those cases which have crept into literature under this heading as possibly being dermoid cysts of the tongue, ranulae or cysts having their inception in the lingual duct and not, as supposed, originating in the anlage of the thyroglossal tract. In the presence of a cyst of any considerable size, it is sometimes extremely difficult to determine the exact location in relation to the thyroid cartilage and thyro-hyoid membrane, for very frequently the swelling will overlap both structures. Consequently, it is very difficult before operation to differentiate suprahyoid, subhyoid, and those in the thyroid cartilage or membrane. It has been almost invariably taught that thyroglossal cysts and fistulae are located in the median line of the neck somewhere along the course of the thyroglossal tract. It is important to bear in mind that this need not necessarily be the case. One of our cases showed a fistulous tract extending to the left suprasternal notch. It is well to bear in mind, however, that both the opening of the fistula or cyst may be eccentrically placed, but the tract itself is almost invariably found in a median position.

The shape of these cysts is often irregular and pouched rather than spherical. Histologically, they are thin walled, lined with either ciliate epithelium or mixed ciliate and squamous epithelium, but seldom squamous epithelium alone. The contents are mucinous, varying from clear yellow to yellowish and opaque. They are never colloid in character, and skin elements are never found, although a certain amount of cornification may be present. Besides the epithelial element noted, a glandular lymphoid tissue may coexist. From the former, multilocular mucinous cysts may be produced, while the lymphoid element may lead to infection from external sources, with the production of granulation tissue and occasional foreign body inclusion.

The fistulous tract may be relatively straight lined, but a frequent characteristic of the fistula is a fluted appearance of the fistulous canal which is ascribed to the collapse and irregular contraction of the thin-walled cyst after it has been emptied. These flutings are cyst processes and do not represent an active extension of the fistulous canal. In the region of the hyoid bone an extensive multilocular cyst may be found and in such cases a considerable irregular excavation of the bone may have taken place.

TREATMENT

The practical importance of this subject is liable to be overlooked until we examine the results and percentage of thyroglossal cysts and fistulae. According to Bailey of London, without the advantage of any special investigation by following up cases, 26% of the total cases operated were proved by the records to be recurrent. A proportion of these had been operated upon twice. It is necessary to make it clear that by recurrent is understood both cases which were operated upon at the London Hospitals and those which originally received similar treatment elsewhere. In view of the intimate exchange of favors among the Hospitals, 26% should represent an even average. In our small series of cases, 33% were operated upon more than once before the cure was obtained.

The following two methods of treatment have given such unsatisfactory results that they have now been largely discarded.

First, the simple dissection of the cyst combined with simple ligation of the thyroglossal pedicle.

Secondly, treatment of thyroglossal fistula by means of scraping and cauterizing.

When an operation for a thyroglossal cyst is undertaken, the goal in view is to extirpate once and for all the cyst and every vestige of the tract. The great majority of thyroglossal cysts are situated in some part of the hyoid portion of the tract. The extirpation of this portion of the tract is comparatively easy. There is nothing to disturb the even tenor of the dissection as far as the hyoid bone. It is here that trouble begins and the conflicting views of leading anatomists upon the relationship of the tract to the subhyoid are forcibly impressed upon the operator. Often it is here that the thyroglossal pedicle, already considerably attenuated, seems to dwindle into a mere shadow and the dissection has to be terminated.

In 1920 Sistrunk reported unsatisfactory results up to date in many of these cases, so he proposed a new operation.

As this is the operation that has given the best results in the treatment of this disease, I will more or less fully present the Sistrunk method.

Should a sinus tract be present it should be injected with a weak solution of methylene blue in order to outline its course definitely. Under general anesthesia, with the patient in the usual position for performing a thyroidectomy, a transverse incision is made over the cyst, through the skin, subcutaneous tissue and platysma muscle. The cyst or sinus tract will be found lying under the raphé adjoining the sternohyoid muscle. This raphé is cut longitudinally and the cyst dissected free from its attachments up to the hyoid bone. The tract usually passes underneath this bone or may pass superficial to it or through it. In order to remove the duct completely at this point a small segment 2-3 m.m. in diameter is removed from the center of the hyoid bone. The mouth is then held open with a gag and the tongue pulled forward with a suture or small stomach forceps. The index finger of the left hand is introduced into the mouth and the foramen caecum located by palpation or inspection. This may be done by following the two rows of circumvallate papillae, one of which lies on each side of the tongue, backward to the point where they join in the median line. The foramen caecum lies just posterior to this point. In doing this it is necessary to bear in mind the direction of the thyroglossal duct. With the patient in the position suggested, this corresponds to a line drawn at an angle of 45 degrees, backward and downward, toward the foramen caecum, through the intersection of lines drawn horizontally and perpendicularly from the center of the hyoid bone; that is, corresponding to a line drawn from the center of the hyoid bone to the foramen caecum.

In temporarily disregarding the duct and coring out the tissues through which it passes lies one of the secrets of cure, as the duct is so small and delicate that it is almost invariably broken, retracts and is lost, if efforts are made to dissect it out alone. The finger in the mouth serves as an excellent guide to prevent entrance through the hyo-epiglottic fold into the mouth or into the pharynx. This is pushed forward and upward with the finger in order to shorten the distance between it and the hyoid bone. Then, without any attempt to isolate the duct, the duct and the tissues surrounding it for a distance of about 3 m.m. on all sides are cored out through the muscles of the tongue to the mucous membrane, at the foramen caecum, where they are cut individually without opening the mucous membrane of the mouth. In dissecting out the duct, a portion of the hyoid bone, portions of the raphé and the mylohyoid muscle, and portions of the geniohyoid and geniohyoglossus muscles are included. The opening through the muscles of the tongue is closed with several interrupted catgut sutures passed superficially through the muscles of the tongue, and the edges of the hyoid bone are brought into apposition with one or two chromic catgut stitches taken through the periosteum of the bone and tissues adjacent to its cut ends. A small rubber tube is introduced deeply into the muscles of the tongue and is allowed to remain for four or five days. The skin wound is closed with interrupted dermal or horsehair sutures.

In regard to our own small series of cases, eight in number, the following data may be of some moment:

There were eight cases, varying from five years to thirty-one years of age, five females and three males, and of these eight cases two needed two operations and one needed four operations before finally cured. These eight cases were all my own, every one at the present time showing complete cure, although the last two cases which I have done are not in this series because they have not been done long enough to warrant any decision.

In conclusion:

1. The embryological development of these cysts shows that they originate from the epithelium of the mouth.
2. To cure these cases, in most instances, an operation must be done which dissects out the fistula from the neck to the foramen caecum.
3. We feel that markedly infected thyroglossal cysts should not be dissected at the first operation through the hyoid bone to the foramen caecum, because of the great danger of the infection running down the mediastinum.

If an operation similar to the one described is performed, most of the cases can be cured.

Discussion

Dr. W. E. Ladd, Boston, Mass.: I have enjoyed Dr. Jarvis' paper for two reasons, one because I think it is a good paper, and the other because I think it is timely, and it is timely because it is high time that we get better results in these cases.

We have had at the Children's Hospital fifty-six cases of thyroglossal duct cysts or sinuses. Of these, twenty-nine have been operated on and followed. Of these twenty-nine, twenty-three have been cured. I don't think that is good enough and I think the causes of the failures and the recurrence are two: (1) not making the correct diagnosis; (2) not doing an adequate operation.

On the diagnosis there should not be very many difficulties and there should be fewer difficulties after a sinus is established than before the cyst has broken through. Almost the only condition that can be confused after a sinus is

established is the bronchiogenic sinus and the position of that is almost always sufficient to differentiate the two conditions.

I think if we all adopt the technique of Sistrunk that Jarvis has described, we will have very much better results than we have had.

In our experience, over a third of these patients had been operated upon one or more times before coming to us, so even though our results are not good, the suggestion is that the results perhaps in other places are even less good. By making a correct diagnosis plus following the technique of Sistrunk, we should have pretty nearly 100 per cent. good results. Our results in the last few years have been very much better than they were previously.

NEW IDEAS FOR PUBLICITY

For several years the American Public Health Association has maintained a Health Education Service which provides "ready-made" material for bulletins, newspaper articles, and the like. A recent addition to this service promises to be of real interest and value to those whose publicity funds are running low. A new photographic process has made possible the transfer of pictures and text to the fiber stencils which are used on duplicating machines. The reproduction of even complicated drawings is remarkable and provides an opportunity for the illustration of mimeographed bulletins.

New uses are constantly being found for this "ready-made" educational material. Some of these are planned by those who are promoting the enterprise and others are devised by ingenious subscribers. One of the features of this service has always been its flexibility, and it appears that more ways of making it adaptable to various purposes are constantly being found.

The ready-to-use plates are now going into numerous state, city and county health department publications, also tuberculosis association bulletins, industrial house organs and school papers. These plates also serve as complete health columns in some newspapers, while the cartoons and pictures alone serve to illustrate local articles in others.

Mats are now supplied in quantities to two state health departments for state-wide newspaper use.

A new process of making stencils for duplicating machines has been adapted to the *Bulletin* service. Text and pictures can now be reproduced with remarkable success on the lowly "Mimeograph." Enlargements are possible up to 8½ inches by 14 inches, opening up new possibilities for making small posters and for using the children's pages in schools.

While not especially designed for children, experience has shown that in places where the *Bulletin* is issued, considerable demand for it has come from teachers of classes in hygiene, civics, biology and the like. The *Bulletin*, under its home name of *American Public Health News*, is now being offered to schools and other organizations in any quantities, when and as desired.

The *Bulletin* continues to be as popular as printed by the Association but appearing under the heading of the local organization putting it out. In New Jersey alone nine health departments are using it each month in this way.

This "Service" appears to be meeting the need for conservative information about personal and public health in an elementary form. The fact that the material is "syndicated" does not seem to lessen its value as there is small chance of the residents of one community seeing the health publications of another. Even if they should, no harm would be done. Six adjoining cities in one locality have used the same pages with evident satisfaction.

There probably has never been another time when "health information" could be given to the public with so much assurance that it would be used. Health organizations have a special responsibility to their people just now. Decreased income—poverty in many cases—is a hardship; combined with illness it becomes a tragedy. Whatever can be said that will help the average family to safeguard the health of its members and to ward off disease is a real piece of public service.

The American Public Health Association is offering a practical solution of this problem of how to get out readable publicity material in an economical way. National and local health organizations have given their whole-hearted endorsement to this easy method of presenting better health material to the public.

Full information may be secured from the American Public Health Association, 450 Seventh Avenue, New York, N. Y.

MORTALITY RATES

Telegraphic returns from 82 cities with a total population of thirty-six million for the week ending October 24, indicate a mortality rate of 10.8 as against a rate of 11.1 for the corresponding week of last year. The highest rate (18.2) appears for Washington, D. C., and the lowest (5.4) for Canton, Ohio. The highest infant mortality rate (17.6) appears for Schenectady, N. Y., and the lowest for Springfield, Mass., which reported no infant mortality.

The annual rate for 82 cities is 11.9 for the forty-three weeks of 1931, and the same rate appears for the corresponding weeks of 1930.—*Bulletin of the Bureau of the Census.*

CARDIOLOGY, OLD AND NEW*

BY WILLIAM H. ROBEY, M.D.†

YOUR committee has asked for a comparison of some of the old beliefs about heart disease with the new. I infer that I am to go back only to the teaching that was given during my days as undergraduate and interne.

Murmurs were talked about a great deal, and, in the minds of many physicians, always pointed to organic cardiac disease. Of course, there were then, as now, functional murmurs, but there was far greater confusion about them than there is today. In fact, physicians were so fearful of making a mistake that they were apt to leave the impression, if not with the patient, at least with his friends, that a "leaking valve" was a serious matter. Sir James Mackenzie was one of the early observers who pointed out the effect upon the child and his family of labeling as organic heart disease a mere functional murmur. It is not uncommon to see in practice today persons who are hale and hearty, who speak with awe and respect of the leaking valve found a generation ago by a trusted physician and carried up to the present time. A leaking valve discovered in childhood, which has permitted its possessor to proceed in perfect health to advanced middle life, should be spoken of with respect. The functional murmur today, while occasionally diagnostically troublesome, has become more readily elucidated since the physician has learned to evaluate the history, together with allied signs in the heart and in the circulation. A murmur which appears in middle or old age as the result of a grave anemia or myocardial hypertrophy is in no sense the result of an endocardial lesion and should be classified as purely functional. To give mitral regurgitation as the cause of death in an old man, as I have known to occur, would be paralleled by giving headache as the cause of death in a case of cerebral tumor.

Far greater clarity in diagnosis, classification, and definition of treatment has occurred in the last twenty years. Diagnosis has been steadily increasing in accuracy and with better understanding of the lesion, or lesions, treatment has shown improvement. There is still considerable laxity in classification, but numerous articles appearing upon the subject and the handbook of the American Heart Association are showing the way to a better interpretation of the underlying causes of heart disease. The background of heart disease must be thoroughly understood if prophylactic and protective treatment are to be intelligently administered. One marked change in the last few years has been the constant effort on the part of investigators to solve the problems of etiology. Declared heart disease must be treated, of course, but the real aim of cardiologists, quite apart from their care of the cardiac cripple, is to prevent the damage which certain focal and acute diseases are capable of exerting upon the various structures of the heart and blood vessels and until these factors are subdued, physicians will continue to lock the door after the horse has gone. Potential heart disease represents one of the greatest problems of present-day internal medicine.

Heart disease today is separated into three main divisions. First, there is the heart disease of early life caused by rheumatic fever; secondly, that of early middle age due to syphilis; and thirdly, that of middle-age and advanced years caused by arteriosclerotic changes.

RHEUMATIC FEVER

Heart disease in the young is one of the manifestations of that infection of varied appearances commonly known as rheumatic fever. Undoubtedly, infections with the organisms of the rheumatic group are responsible for much of the cardiac disability of the third, fourth, and possibly the fifth decades. Yet in my student days when many cases of rheumatic fever could be seen in the wards, it was looked upon as a systemic disease with acute joint manifestations. I do not remember that any attention was paid to foci of infection. Diseased tonsils, sinuses, teeth and any other possible areas which might harbor infecting bacteria were allowed to accompany the patient upon his discharge from the hospital. Sir Andrew Clarke, upon being asked what he considered the best treatment for rheumatic fever, replied, "Six weeks." We gave the patients that form of treatment with complete rest in bed, a light and easily digested diet and large doses of salicylate of soda. At the end of six weeks, the average patient was ready to leave the hospital and we stood ready to readmit him, which we often did. Common causes for acute rheumatism were considered to be getting wet, chilled or overworked. The rest raised the resistance of the patient until he established a temporary immunity to the infecting agent. A few weeks or months after he resumed his occupation, the infecting focus overcame his resistance and he returned to the hospital with another attack.

Chorea was looked upon as an entity and was treated as such, the drug used being arsenic and never, so far as I know, salicylate of soda. It was considered an affection of the nervous system and quite unrelated to rheumatism. It could produce a valvular lesion, just as rheumatic fever, and yet it was never classed as an infec-

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tion. Similarly we went through the same process of raising the patient's resistance and seeing the return of chorea when the resistance was lowered. As in rheumatic fever, foci of infection were never considered. Our visiting physician at the Children's Hospital noted that chorea occurred in conscientious school children more often in the fall of the year, when such children were adapting themselves to new school work and in the spring when they were preparing for final examinations. He also distinguished true chorea from choreic twitchings because the former was often accompanied by a mitral systolic murmur, and when such a murmur was discovered, he unhesitatingly made a diagnosis of chorea. Yet the appearance of an endocardial lesion never suggested a blood stream infection. In treatises on general medicine today, chorea has not disappeared as an entity, but is described as an acute infectious disease characterized by involuntary, coarse muscular twitching, psychical changes, and a tendency to endocarditis and polyarthritis. A non-hemolytic streptococcus is believed to be the exciting agent. Huddleson in Cecil's *Practice*, 1930[2], makes the interesting statement that rheumatism *per se* is not really a cause of chorea but is rather part of a multiple symptom complex (rheumatism, endocarditis, chorea) caused by a specific virus. This fact has been known to physicians in large hospital clinics for many years. Even more striking is the diversity of arrangement and the priority of onset of the several symptoms of the complex. In one child it may be a tonsillitis and chorea, or the order may be reversed, or an articular inflammation may precede the chorea to be followed by an endocarditis, which is often the case.

A polyarthritis with fever involving the smaller joints, such as the fingers, and the larger ones, as the knees and ankles, very painful on motion, hot, red and tender to the touch, is familiar to all clinicians and need not be more fully described; if, however, we are to depend upon such a manifestation of the disease, especially in children, we will make many failures in diagnosis. Rheumatic fever under two years of age has been authentically reported, but it is very rare. In children the joint symptoms are of much slighter character while the cardiac lesions are much more pronounced and frequent. Growing pains, or pain in the thigh or calf, should be taken as seriously as pain and swelling of the joints. Vague sore throats, nervousness and irritability quite apart from chorea should never be passed over lightly. Unexplained fever, gastric pain, nausea, vomiting or epistaxis may all be indicative of rheumatism in a child. A few days ago I saw a boy of eleven whose physician stated that one year ago he had an illness characterized by fever, sore throat and double cardiac murmurs. He recovered and had been reasonably well, but upon questioning his family, I found that he had not been able during the last year to play so vigorously as the other boys and that he seemed rather easily fatigued. His physician said that following recovery from the previous illness, his murmurs had disappeared. Upon physical examination the temperature was 101°, the pulse 100 and the respirations slightly increased. The tonsils were small and the right one was easily seen although tonsillectomy had been performed when the boy was five years old. The heart was normal in size with a loud to-and-fro friction rub over the entire precordia. The cardiac impulse was strong and heaving. In the area just below the angle of the left scapula, there was dullness with slight increase in the voice sounds suggesting Ewart's sign of a posterior pericardial effusion. The lungs were otherwise clear. The carotids pulsated violently. The joints were normal. When I made a diagnosis of rheumatic fever with pericarditis, the physician asked me how I was justified in making a diagnosis of rheumatic fever since there was no involvement of the joints. I explained to him that the evident attack of tonsillitis followed by pericarditis was sufficient without swollen tender joints especially since children, unlike adults, often did not have them. Were there murmurs hidden behind the loud, pericardial rub, or was the previous attack one with only a pericardial rub? If the boy survives the present inflammatory process, it remains to be seen what endocardial lesions exist. Even a transient pericardial rub heard for a brief time during rheumatic fever in a heart which at the end of the fever is apparently free from endocardial lesions should make us cautious in our prognosis and eager to follow the subsequent course, remembering that inflammatory processes are not apt to select any one structure of the heart but are likely to attack with varying intensity all of its tissues.

The large majority of damaged hearts in the young result from involvement with the rheumatic group. Other varieties of infectious chronic valvular disease are less common for the reason that, when such organisms as the pneumococcus, gonococcus and meningococcus attack the endocardium, they do so as a part of an overwhelming toxemia from which the patient succumbs. In a study of pneumonia in dogs the writer, working in conjunction with Dr. H. T. Karsner, found that only in animals infected with overwhelming doses of a virulent pneumococcus was there any involvement of the heart. It is a common finding that, if a patient survives even a severe pneumonia, the heart is usually found to be unharmed. Pneumococcus endocarditis occurs, but it is rare. Reports indicate that the gonococcus, when it attacks the endocardium, as it rarely does, is usually a fatal bloodstream infection and has a predilection for the pulmonic valve. In such a case seen by the writer in a soldier during the war, the diagnosis of gonococcus pulmonic stenosis was made and proved

by autopsy. The physical signs were probably produced partly by inflammatory and partly by edematous alterations in the tissues.

Rheumatic fever, while often a prolonged and painful affection entailing considerable economic loss, could be borne in face of the above obstacles were it not for its crippling cardiac complications, which occur in about 80% of the cases. In fact, manifestation of toxemia and edema of the cardiac tissues occurs in 95% of the cases during the height of the attack. Complete heart block was observed as a transient complication in 50% of our cases.

The most outstanding cardiac complication of rheumatic fever is an inflammation of the valve leaflets, chordae and adjacent tissues, resulting eventually in cicatrization which interferes with the normal passage of blood through the auriculo-ventricular opening, producing the mechanical defect which we call mitral stenosis. If there is a warping of the valve leaflets, it is reasonable to suppose that they can no longer approximate accurately and hence blood will regurgitate into the auricle when the ventricle contracts upon its complement of blood. It is also evident that if an inflammatory process causes a change in the shape of the valve ring, the cusps can no longer approximate and regurgitation is sure to occur when the ventricle contracts. Accurate and detailed descriptions of mitral regurgitation and stenosis are given in all the modern textbooks on diseases of the heart so that I will take the time merely to discuss certain controversial points. A generation ago, mitral regurgitation in the young was an often-made diagnosis, but today a systolic murmur at the mitral area, especially if it is loud, blowing or rough, masks all or a portion of the first sound, is accompanied by a systolic thrill, and is transmitted well into the axilla and possibly the back. With a history of rheumatic fever, chorea or tonsillitis and some enlargement of the heart, a loud systolic murmur at the apex is almost certain to indicate damage to the mitral valve, provided there are not present causes of cardiac dilatation, such as the functional regurgitation which one sees so much oftener in the enlarged hearts of elderly people, anemia, debilitating diseases or cardiac failure. In older people with cardiac enlargement due to arteriosclerosis the murmur of functional regurgitation is often softer and less apt to be accompanied by a thrill, than is the case of a valve lesion. Formerly a functional systolic murmur at the apex in a person of any age was unable to assure the physician of its innocence and he was inclined, merely upon the strength of an abnormal blowing murmur, to make a diagnosis of valvular disease. When the thrill, and especially the rough systolic murmur, are persistent, particularly with a history of any of the symptoms of rheumatism and cardiac enlargement in a young person, we should make a diagnosis of rheumatic heart disease, but we should be on the watch for the advent of mitral stenosis. In my student days, the diagnosis of mitral stenosis was rarely made in the absence of a presystolic crescendo murmur and, in addition, the examiner was keen to discover a thrill, over which he spent much time in attempting to prove that it was also presystolic in time. Doubtless mistakes were often made because a short presystolic murmur and thrill may be found in nervous hearts beating forcefully as well as in mitral stenosis. I made this mistake occasionally in my early days and especially when I first began to see cases of neurocirculatory asthenia.

Today we recognize the possibility of six different murmurs in rheumatic mitral heart disease: the regurgitant mitral systolic, the middiastolic, the presystolic, the early diastolic, the late diastolic and the full diastolic. The presystolic crescendo murmur is the most positive evidence of mitral valve disease at our command, yet the presystolic murmur without the soft blowing middiastolic murmur is unusual. The blowing diastolic murmur of the early, late or full type depends upon the strength of auricular systole, the heart rate and the degree of stenosis. Changes in rhythm and rate, so apt to occur in advancing mitral stenosis, such as auricular fibrillation or paroxysmal tachycardia, usually dissipate the presystolic murmur and may, or may not, cause a disappearance of the middiastolic. Auricular fibrillation does not always cause cessation of the presystolic murmur, as has been pointed out by Lewis. The middiastolic, however, is more persistent and is a much safer diagnostic sign than the presystolic. The diastolic murmur always follows the second sound by a brief interval differing from the diastolic murmur of aortic regurgitation in which the second sound of the heart is almost invariably lost. Furthermore, in the latter lesion, there are the characteristic peripheral circulatory signs. The appearance of auricular fibrillation or paroxysmal tachycardia is strongly suggestive of organic mitral disease in a young person who has been carrying a systolic murmur or a doubtful middiastolic.

During my interne days the treatment of rheumatic fever was the use of salicylate of soda, soothing applications to the inflamed joints and absolute rest in bed until two or three weeks after the subsidence of the fever and until we felt that resistance had been raised. While this treatment was being carried out, we sat patiently by hoping that the heart would somehow escape injury. We all know how futile that hope often was. We are no better off today in many ways, but we do appreciate that if we are to end the crippling cardiac disease of youth, we must eradicate rheumatic fever. Banish or reduce rheumatic fever and you will prevent endocarditis in youth and in those who escape endocarditis, rheumatic myocardial de-

generation in middle life due to fibrotic changes. Various forms of streptococci sera have recently been tried with not very satisfactory results. Homer Swift and his associates are now using a serum but state that it is too early to come to any definite conclusion. In a few patients with acute pericarditis, serum therapy, combined with anti-rheumatic drugs, has led to a more rapid recovery than would have been anticipated from drugs alone.

Recently the work of Charles C. Johnson by animal experimentation has shown that the use of bicarbonate of soda, together with salicylates in full therapeutic doses, in rheumatic fever is rational. The bicarbonate for a long time has been used empirically in doses twice the size of each dose of salicylate to counteract acidosis and to prevent gastric irritation.

While we are waiting for the discovery of the specific organism and serum we must give more thought to the foci of infection. Even after the organism has been found, we will still be faced with the problem of the focus in each case. It is a well-known principle that if a focus can be removed surgically, the bloodstream will often free itself of a bacteriemia without assistance, but if, as in a case of subacute bacterial endocarditis, the focus cannot be reached, vaccines are completely dissipated by the constantly active focus.

TONSILS

In most articles on rheumatic heart disease the tonsils are placed first in the list of causes; yet when various authors discuss this subject, they express grave doubt about the value of tonsillectomy, claiming that the operation does not prevent subsequent attacks of rheumatic fever. Those who believe in the enucleation of the tonsils as a preventive think that the reasons for failure may be classed as follows:

(a) The operation may be imperfectly performed and tonsillar remains be left, which act as a focus in subsequent attacks. Robey and Freedman, in a study of 454 cases of rheumatic fever, found that of 201 tonsillectomized patients, 139, or 31 per cent., did not develop subsequent rheumatism or chorea; 45, or 10 per cent., had subsequent rheumatism, and 17, or 4 per cent., had subsequent chorea. In this group there were 32 patients with the evidences of poor tonsillectomy.

(b) If the tonsils are allowed to remain through repeated attacks, they succeed in establishing other foci, principally in the synovial membranes of the joints, which, in turn, act as foci of infection, even though there has been a complete tonsillectomy.

Kaiser found rheumatic heart disease in 450 of 20,000 tonsillectomized children and in 817 of 28,000 who were not operated upon. Many of the children in the former group developed heart disease before tonsil enucleation. A careful analysis of 478 cases of carditis showed that in 83 per cent. the condition developed before tonsil removal and in 17 per cent. following tonsillectomy. He concludes that the tonsillectomized child is assured a greater protection against rheumatic fever, scarlet fever and chronic heart disease than the one who has not had his tonsils removed.

Robey and Finland studied 165 cases of rheumatic fever: 71 patients were operated upon for infected tonsils, while 94 were used as controls. They concluded that rheumatic fever could be brought to an end after study and elimination of other foci by performing tonsillectomy, even during the height of the fever and inflamed joints. The patients were not operated upon until all of the usual medical measures had been thoroughly tried without success. The object of the operation at this time is to save the heart, if possible, by ending the infection, and to reduce economic loss. In 16 the results were brilliant, in some the entire process being brought to an end within three days, when it had been going on with repeated attacks from four to ten weeks. In 8 the operation apparently accelerated improvement; in 9 there was no apparent effect on the course. No deaths or complications occurred in the operated cases and they recommend it as a perfectly safe procedure when other methods fail. In 21 cases operated upon while quiescent, 4 had a mild return of symptoms. Four had a moderate return, 1 had severe sore throat and 1 a postoperative hemorrhage. In the 94 unoperated cases there were 3 deaths; 2 due to pneumonia and 1 to pulmonary infarction. Laryngologists are still fearful of precipitating a general bloodstream infection with death by operation during an active rheumatic infection. This did not occur during our series. On the other hand, the writer has seen two deaths in private practice within a year from tonsillitis without operation.

Since these reports appeared, a young man of twenty-one, the brother of a surgeon, was seen in one of our private hospitals. He had a polyarthritis with the usual hot, tender swollen joints and fever following a tonsillitis. After a thorough course of salicylates lasting some three weeks during which a cessation of symptoms alternated with recurrences of acute joints, all foci were eliminated except the tonsils and two or three of our best laryngologists discussed the problem of tonsillectomy but expressed the fear of inducing a fatal septicemia. At last one of them consented to perform the operation, which was successfully done, with the result that within four or five days, the joint inflammation entirely ceased and over a year later the patient's brother told me that he had been perfectly well since. Fortunately, so far as could be discovered, there was no cardiac in-

volvement, yet while the polyarthritis was going on, the attending physicians sat anxiously and helplessly by, hoping that the heart would escape.

TEETH

The teeth are less apt to cause cardiac involvement than the tonsils. In the writer's experience, healthy-looking teeth are more frequently a source of obscure cardiac infections than the clearly diseased ones. By this we mean that a firmly embedded tooth with an infected root canal is a greater source of danger than a decayed tooth loosely placed in the alveolar process and evident to the patient and his dentist. That infected teeth occasionally are a cause of cardiac pain cannot be doubted. Usually x-ray studies and their competent interpretation are necessary to determine the possibility of root canal infection.

SYPHILIS

Next to rheumatic fever as a cause of heart disease, especially in the young adult, comes syphilis.

The term "syphilis" was first used by the distinguished Italian physician, Fracastoro, in 1530. At the end of the fifteenth century an epidemic of great intensity invaded Europe. Charles VIII of France entered Italy in 1494 and 1495 with an army of adventurers from various countries accompanied by a dissolute train of camp followers. A venereal plague ran through the Italian cities. It was at this time extremely malignant, a fact explained by Bloch, who maintains that this was the first appearance of the disease in the Old World because it was implanted on virgin soil. The secondary symptoms appeared very early; often in a few days there was high fever, intense pains in the joints, severe skin affections, and frequently death. It is said that the comparative mildness of syphilis today is due to the fierceness of this epidemic.

Las Casas, who lived for many years in the Island of Hispaniola, questioned the Indians and was convinced that the disease had long existed among them but in a far milder degree than among Europeans. Columbus returned from the West Indies, in 1493, bringing with him ten Indians from Hispaniola to Seville, who, together with a few infected sailors, spread the disease through Spain. It is interesting that syphilis was so mild in the West Indies, where it had existed for so many years that the virus had become much attenuated. With the Europeans, unaccustomed to the disease, it played havoc. In like manner, during the World War, measles attacked with considerable violence the soldiers from the Virginia and Kentucky mountains, unaccustomed to contagious diseases.

The French spoke of syphilis as "mal napolitain" and the Italians as "mal francese"—neither seemed to covet the honor of its introduction. According to Oviedo and later, Osler, syphilis was an American disease. No European bones earlier than 1493 showed evidence of it.

In the eighteenth century Lancisi described cardiac syphilis and supported the observations of Paré, who considered syphilis as one of the causes of aneurism.

It is generally agreed that syphilis is essentially a disease of the blood vessels. From the beginning of the initial lesion to the final termination there is always endarteritis and periarteritis.

It is evident then that in almost any clinical manifestation of syphilis a vascular element is present. No matter where it occurs, whether it be in the gumma, the syphilitic gastric ulcer, the smooth serum discharging surface of the chancre, the hemiplegia due to rupture or thrombosis in the internal capsule, or angina pectoris due to syphilitic coronary disease.

Knowing, then, this evident fact that the spirocheta treponema pallidum tends to involve the vascular system in practically every patient, it is our duty to remember that no small part of syphilis of the vascular system will appear in our practice under the clinical mask of diseases of other structures but may fail to present any definite symptoms. Vascular changes may be recognized, as in syphilis of the nervous system, by certain signs and symptoms, but, to quote Stokes, it may always be assumed that when one-tenth of the process is causing symptoms which come to clinical recognition, nine-tenths lie beyond the range of our clinical vision.

HISTORY OF SYPHILIS

Let us look critically at the medical history of our patients as they come to consult us and let us assume that they are men, since three-fourths of the cases of cardiovascular syphilis occur in them, not forgetting, however, the remaining fourth where the establishment of the diagnosis is even more difficult.

A considerable number of men will admit frankly an attack of gonorrhea, especially if it occurred during the early years before they settled down to the respectability of married life. In Stokes' series one-third of the patients with syphilitic cardiovascular disease could give no history pointing to infection. It might have been masked by the discomforts of gonorrhea or forgotten in the long interval between the initial lesion and the onset of the vascular symptoms. Every one of us has had experiences such as I will relate.

A patient whom I had known very well for many years, upon whom I had made numerous physical examinations and to whom I had talked

about the dangers of allowing syphilis to go on year by year without renewal of treatment, came to me, one day, with an eruption which had much the appearance of psoriasis. The idea that this very correct and highly respected man was syphilitic did not enter my mind and I treated his psoriasis for a time because I had always been his physician and he did not wish to go to a dermatologist. Finding that the psoriasis was intractable, we finally consulted with a dermatologist, who said that, because of the tiny white scars which some of the healed lesions had left, it was undoubtedly a psoriasiform late syphilid. The blood and spinal fluid Wassermann tests were negative. This man, although nearly fifty years of age, went for twenty-eight years without antisyphilitic treatment, in fact, no treatment since that given for four or five months immediately following the appearance of the initial lesion. He was most fortunate in the mildness of his infection and we may say of his spirocheta pallida what Lawrason Brown once said of the tubercle bacilli in a patient of mine who went to Saranac, that his bacilli had been to Sunday School. No history of a sore had ever been obtained from this patient and he had kept his early experience a secret until confronted with the statement that his skin lesions were syphilitic. Under several treatments with arsphenamine his skin quickly cleared and there have been no subsequent manifestations.

I acquired some wisdom from this experience, but not enough. A few years later a very active man of sixty-one, on whom I had made careful routine physical examinations for a few years and who had always been in perfect health, developed a small sore on the arm, which failed to heal. A blood Wassermann test was positive and when told of the result, he said that he had a small sore at the age of sixteen, the meaning of which he had never understood. So far as he could remember, he had never received any treatment for it. Although he has received vigorous antisyphilitic treatment, his blood Wassermann reaction remains positive. Yet I have never been able to discover any blood vessel lesions which would suggest vascular syphilis.

The virulence of the spirocheta pallida must, unquestionably, vary, as does that of all other parasites and bacteria. Again, the differentiation of the spirocheta pallida into sub-varieties or strains is becoming more and more recognized. The presence of syphilis in husband and wife infected by the same organism tends towards the same clinical manifestations. The well-known series of cases in which five army officers infected by the same woman all developed paresis is another clinical example of the selective localization of a certain strain of the parasite. Since involvement of the vascular system is so common, it must be that a variety of strains attack this vital tissue.

Fordyce says: "It cannot be emphasized too frequently nor too emphatically that the fate of the syphilitic individual depends largely upon the early diagnosis of his infection and the intensity with which his treatment is carried out in the first six months. It is in the accomplishment of this purpose that the modern aids to diagnosis have rendered such invaluable service."

The diagnosis of syphilis should be made at the earliest possible moment by use of the dark-field illumination and the Wassermann or its allied tests.

From my consulting-room experience, I can say that a history of gonorrhea is frankly admitted by a large number of patients who have no knowledge of a possible syphilitic infection. It is a good plan to examine the blood of all your patients for the Wassermann reaction, but especially any who admit that they have had gonorrhea. The technique is simple and the expense nil since most Board of Health laboratories are equipped to perform the test.

Stokes, in speaking of the vascular distribution of syphilis, aptly says that the infection may be ubiquitous, insidious or disastrous. It is, therefore, necessary, knowing its tendency to invade most organs, and especially the vascular system, to be ever on the watch for syphilis. When a man of character or position, who gives no history of a syphilitic infection, presents himself with vague or undefined symptoms, or with any of the known signs of blood vessel or cardiac degeneration, we should suspect its presence and apply our tests. With such persons we are sometimes inclined to dismiss syphilis with the thought that their evident integrity is against it, but, knowing as we do that human nature is always the same, as pointed out by Voltaire and others, and that man will seek to gratify hunger and sexual desire, it may be that some time in the dim past, the most upright man may have had the misfortune to acquire the infection.

Usually the disastrous period of vascular involvement is the tertiary stage and even then, although diagnosis is more evident, early discovery is essential since treatment may be of some, though far lessened, service. Sometimes the initial infection is so violent that the usual sequence of stages is disturbed, as Brooks and later Reid have pointed out, so that a heart may become seriously involved a few weeks later. Brooks has reported a case in which death resulted from perforation of the aorta just above the valve before the secondary eruption was fully developed and Reid saw a case at the Massachusetts General Hospital of a young man infected in June, who had sufficient signs for a positive diagnosis of syphilitic aortitis in September. Fortunately, such cases are not common, but they occasionally occur, unhappily for the patient and his physician. Usually the tertiary manifestations of aortitis come on twelve

to twenty years after the initial lesion. During the first year or two of my practice, I treated a young man for a chancre, during the absence of an older colleague. A little later he was returned to my friend and I did not see nor hear of the patient until fourteen years afterwards, when I saw him in one of the wards of the hospital, with a well-marked aortic regurgitation. He denied ever having had syphilis, but when I reminded him that I had treated his chancre, he sheepishly admitted the facts. The average time for the appearance of syphilitic aortitis is about fifteen years after infection. Thus it so often happens that in early middle age, when the man is entering upon the benefits of a successful career, he is halted by a crippling disorder. Cardiovascular syphilis is seldom recognized in the first decade, forty-five per cent. of the cases are recognized in the second decade, and thirty per cent. in the third. Aortitis precedes aneurism pathologically and chronologically. Many of our cases never go on to aneurism, the patient dying of cardiac and circulatory failure before the former condition is reached, or at least before it is possible to recognize it clinically. Valvular damage causes more pronounced symptoms than aneurism or even myocardial degeneration. On the other hand, an aneurism must show its presence by pressure and therefore must have size or position before its signs are evident to the average examiner.

Since the introduction of the Wassermann reaction and its allies, we have been greatly helped, yet it is sometimes absent when we fully expect it to be positive and positive in some cases to our great surprise. One negative reaction, however, should not decide against a diagnosis of syphilis; in some cases as many as five or seven tests are necessary before a positive reaction is obtained. Longcope found the Wassermann reaction positive in 25 to 68 per cent. of cases of syphilitic vascular disease; 75 to 80 per cent. in aortic insufficiency; 85 to 90 per cent. in aneurism; and 75 to 88 per cent. in general aortic disease. In some cases a positive reaction is provoked by mercury or arsphenamine.

The frequency with which neurosyphilis and vascular syphilis are combined in the same patient has led Stokes to say that a routine neurological examination and spinal fluid examination should become parts of the study of every syphilitic patient with cardiovascular disease. On the other hand, it should be remembered that the presence of neurosyphilis does not necessarily prove that the vascular disease is of syphilitic origin, although I believe that we should give the patient the benefit of the doubt. I am afraid that many of us have not reached the point of testing the spinal fluid in every suspected case of vascular syphilis, although Frothingham called attention, a number of years ago, to the striking results of testing the spinal fluid in a series of patients, at the Peter Bent Brigham Hospital, who presented vague symptoms of previously unsuspected origin. The technique is much more difficult than for the blood reaction.

In reading books on the physical signs of aortitis, myocarditis and coronary disease, the diagnosis of the syphilitic origin seems simple, but from a long experience with these conditions, I believe it is extremely difficult to separate these cases into the syphilitic and nonsyphilitic. It becomes easier when we are dealing with patients under forty-five. The early use of the Wassermann reaction as a part of our general examination in every case, not only of those with cardiovascular disease but of our other patients, will serve to prevent the signs which may surprise us after all too obvious damage to heart and blood vessels has occurred. To diagnose a widened arch, a loud blowing diastolic murmur in a heart with its impulse in the sixth space and a water hammer pulse, to feel a pulsating substernal tumor, or discover that a young or middle-aged man has coronary disease because of dyspnea and precordial distress, are interesting findings for a medical undergraduate, but for the practicing physician, they are merely the signs of helpless medical mediocrity. Nor should we be lulled into a negation of syphilis in a young man because he has what the history suggests, a tonsillar or rheumatic infection with pains in the joints and a resulting aortic lesion, possibly without a complicating mitral involvement. There may be a negative history of syphilis and we are contented with our one negative Wassermann test. While all cases of aortic valvular disease do not come from syphilis, still, of all the infections syphilis comes first in the production of uncomplicated aortic valvular disease with aortitis. We have been taught, and still teach, that aortic stenosis is due to some infection other than syphilis, but have we adequate and convincing knowledge that this is true? We feel satisfied that mitral disease is due to the streptococcus or its allies and never to the spirocheta pallida. Warthin stated that he had never found a case from this source.

Arteriosclerosis in a young person should always make us suspicious of syphilis, while in the old, signs of insufficiency, dilatation of the arch, and even aneurism do not necessarily mean syphilis. Within a year, however, a man of 76 years was admitted to my service with aneurism of the ascending arch and a strongly positive Wassermann reaction. He denied an initial infection, so that we do not know how long the disease had existed. He was by far the oldest patient with syphilitic aortitis and aneurism I have ever seen. In your doubtful cases of arteriosclerosis in young persons, you may provoke a Wassermann reaction by the use of mercury and iodide of potassium for about six weeks, to be followed, if necessary, by four to six weekly injections of arsphenamine. I do not believe the

reverse order should be used for fear of inducing the Herxheimer reaction.

ARRHYTHMIA

The greatest advance in cardiological interpretation in the last two decades has occurred in the arrhythmias. This has been due to the electrocardiograph in a large measure. Until Einthoven's discovery some of the arrhythmias were recognized but their relative importance could not be estimated. The use of the instrument has made experimental investigation possible and the correlation of definite pathological findings in the human heart. While arrhythmia may occur at any age, its greatest significance is in the middle-aged and old heart. Without an understanding of the arrhythmias the physician of today is at a loss in diagnosis, treatment and prognosis. Unfortunately for us, the electrocardiograph is not infallible, for serious disease may exist and death occur without a sign from the instrument. On the other hand, it sometimes shows cardiac conditions, such as bundle branch block, which are totally unsuspected by the clinician. The electrocardiograph is an instrument of great delicacy, rather simple to operate, but requiring special training to interpret its various photographic curves. While a valuable adjunct, it can never replace a competent history, physical examination and one's common sense. It has so awed certain clinicians in the past that they have seemed to throw away their training and experience in an attitude of helplessness.

Premature contractions represent the commonest form of arrhythmia. If they begin in youth, they are often harmless; but if they first appear in middle life, especially if accompanied by any other signs of cardiac degeneration, they are of some importance. If the premature contraction is of sufficient force to open the aortic valve, it will result in a radial wave, but if not of sufficient force, there will be a fainter impulse at the apex area and absence of the radial wave. The long compensatory pause while the ventricle is waiting for the next regular auricular impulse is what the patient notices and if unusually long, it annoys and sometimes frightens him. Many patients are, however, quite unaware of the presence of premature contractions. Dropped beats, one of the earliest manifestations of heart block, may be distinguished from premature beats by the simultaneous absence of cardiac impulse and radial wave. Digitalis in 1½ grain doses, three times daily, or quinidine, 3 grains three times a day, sometimes has a favorable effect on premature contractions, but quite as often these drugs make little difference. Bromides have, perhaps, the best action. Some people are very susceptible to coffee or tobacco and stopping their use not infrequently dissipates the premature contractions. In many cases a frank explanation of the extrasystoles reassures the patient and he ceases to worry about them, which is helpful. In young people they are sometimes present as a symptom of mitral stenosis.

Tachycardia, years ago, was considered a purely nervous phenomenon occurring in "high-strung" people of any age. For some time, however, it has been looked upon as a manifestation of abnormal mechanism in which the normal pacemaker fails to function or is submerged in the temporarily rapid rhythm, or in other cases there is a loss of vagal control. A variety of factors seem to enter into its production, such as tobacco, fatigue, indigestion, digitalis, alcohol, infections and heart disease. I have known patients who, if they indulge too much in strong coffee or cigars, especially in the evening, will awake in the night with a tachycardia. Usually attacks last but a few minutes. When they first appear, they are alarming, but we are always able to reassure our patients and often they become accustomed to the attacks. Occasionally we see cases where the attacks come at lessened intervals and finally, after a number of years, there is an attack which cannot be stopped, the patient passing into the stage of complete heart failure and death. One of our worries is caused by the question of just how seriously we shall regard the attacks. We know that in young persons they are sometimes an early manifestation of rheumatic heart disease. Why in the middle-aged does one person have from over-indulgence in tobacco an attack of paroxysmal tachycardia while another has no disturbance? Does it mean as in some cases of premature contractions that there is an organic basis? Often there is no need of treatment, especially when the attacks are brief in duration and of infrequent occurrence. Rest for some, lying or sitting, exercise for others, such as moderate calisthenics, dissipates the attack. People learn to assume certain positions which help. Sometimes if the physician is at hand, pressure on the jugular bulb or the eyeballs for fifteen or twenty seconds may cause cessation. Drugs are of little value. In prolonged and more distressing attacks quinidine sulphate or digitalis, or both, may be tried. In the prevention of attacks these drugs have been helpful in about one-half the cases.

In one case in the Boston City Hospital, this winter, digitalis and quinidine failed, but quinine hydrochloride, 10 grains every 4 hours until the patient had received 80 grains, reduced the heart rate promptly from 176 to 80 beats. The rate remained down but the patient reëntered a few months later with coronary thrombosis and died. The persistent tachycardia was merely an earlier manifestation of cardiac degeneration. Persistent tachycardia of low rates should suggest the possibility of hyperthyroidism. Estimation of the basal metabolic rate is the best guide.

Auricular flutter is a regular increase in rate which may be mistaken for tachycardia, as sometimes happens when an attack of tachycardia

lasts for several days. A tachycardia persisting for more than two weeks should make us consider the possibility of auricular flutter. The rate of flutter is never so high as that of paroxysmal tachycardia, 130-150 on the average, as compared with 180 or 200. Furthermore, simple paroxysmal tachycardia usually lasts not more than two weeks while flutter may exist for months or even years. The ventricle is protected from overwork in flutter by a 2-1 block although any grade of block may be present. While the rhythm of flutter is surprisingly regular, it may be temporarily irregular while the grade of block is changing from one to another. The treatment of auricular flutter is digitalis.

Auricular fibrillation is well known. It is the most disordered action of the heart known and occurs in rheumatic heart disease, hyperthyroidism and degenerative diseases of the myocardium. A thorough search for any of these etiological causes should be made in any case. Low rates of fibrillation are sometimes difficult to distinguish from premature contractions and here the electrocardiograph gives assistance. The treatment is digitalis and quinidine sulphate. Digitalis does not affect the auricular time but spares the work of the ventricle by blocking the impulses from auricle to ventricle. Because it did not affect the rapid action of the auricle, quinidine was welcomed about fifteen years ago. It is a dangerous drug in cases of long-standing auricular fibrillation and deaths have occurred from its use even in small doses. Slowing the auricular time may dislodge a minute portion of the thrombus which a rapid and imperfect action of the auricle has allowed to accumulate. Deaths from cerebral embolus have thus been brought about. In early auricular fibrillation the drug may be used, beginning cautiously, with the patient preferably in a hospital, by giving an initial dose of three grains and then waiting to see if any of the symptoms of quinine poisoning are produced. If not, the drug may then be given intensively, as much as six grains every two hours until thirty are reached, and so continued for ten days if the electrocardiograph does not show a return to normal auricular rhythm before that time.

Block in its varying degrees may be recognized by the lowered heart rate, complete block giving sometimes a rate as low as twenty-eight beats per minute. The electrocardiograph assists in determining the degree of block. Many drugs have been tried to relieve the condition but without much success. In the Stokes-Adams syndrome of complete block we are forced to prevent, if possible, the alarming, convulsive seizures with brief loss of consciousness. Atropine sulphate will do this sometimes by paralyzing vagal control. More recently an old cardiac drug, barium chloride, has been tried (gr. j 3 or 4 times daily) with some success in the relief of the symptoms.

Alternation of the pulse is a serious and little understood phenomenon occurring in degenerated hearts. You can best discover it with your aneroid blood pressure apparatus.

Angina pectoris and coronary sclerosis have become more and more clarified in the last few years. They provide the material for many lectures and cannot, in our brief time, be more than mentioned. The former is a manifestation usually of organic disease of the coronary artery. It is a symptom and not a disease. Frequently there are other signs of a degenerated heart, but occasionally they are entirely lacking. Severe attacks and even death may occur without clinical or instrumental findings. I once saw an apparently healthy man complaining of severe nocturnal seizures with sweating, who died in about six weeks from coronary thrombosis. He was seen by several physicians, two of them cardiologists, but none could find an abnormal sign.

Let me remind you of the importance of nocturnal pain and attacks of dyspnea in older hearts. Both suggest the possibility of a degenerated heart. They occur when the heart slows during sleep. Sometimes angina pectoris exists for years, then comes an attack of coronary thrombosis. If a large branch of the coronary is involved, death may be instantaneous. Infarctions of less extent produce violent pain, collapse, thready pulse, sweating, vomiting, pericardial friction rub, fever and moderate leucocytosis. The pain of angina pectoris is often relieved by the nitrites, but that of infarction never, several doses of morphia being often necessary. After an attack of angina pectoris, the patient is soon about, but with coronary thrombosis he must be kept in bed from four to six weeks.

Lastly, I can recall seeing cases of sinus arrhythmia in young persons when I was beginning practice. After taking a thorough history and making a complete physical examination, I assured their parents that the arrhythmia was a matter of no consequence, although I must say I was a little nervous about my statement. It is a normal respiratory phenomenon occurring in children and sometimes young adults. In the elderly heart with sinoauricular bradycardia with which sinus arrhythmia may be associated, it is an important condition, resulting sometimes in syncope and sometimes death.

This is, at best, the briefest sketch of some of the arrhythmias. Full and excellent descriptions of them are to be found in all of the modern works on clinical medicine, especially those dealing with the heart. The mechanism of the arrhythmias is clearly given in a very readable book by Sir Thomas Lewis: "The Clinical Disorders of the Heart Beat."

Those of us here tonight who graduated in Medicine before the beginning of the twentieth

century have seen marked changes in the preparation and use of digitalis. Through the efforts of physicians and pharmacists impotent tinctures have been disappearing from the shelves of drug stores. In place of the tinctures, which are not entirely out of use, many excellent pills and tablets of the dried leaf have appeared. These tablets made from carefully prepared digitalis leaves are accurately weighed and sold in small phials. If left for the patient to take, the physician knows exactly what dose he is receiving in twenty-four hours. The uncertainty of measuring a liquid leaves him in doubt. The physician has gradually overcome his dread of giving anything but tiny doses for fear of the cumulative effect of the drug. He has learned that large doses in fairly quick succession must be given if he is to steady a failing heart and he has also learned that after the digitalization of his patient, he must estimate what the maintenance dose is to be. Often in the past we made the mistake of not following up our digitalization with a sufficient dosage to prevent a relapse. Also in giving a digitalis tincture we have learned that the drop varies greatly in size and that it requires at least two drops to make a minim. Even the measuring of a minim may be faulty. Personally I have not used a tincture outside of the hospital clinic for many years. The physician has also learned that, apart from headache and gastro-intestinal disturbances, digitalis may poison the heart, especially if given in excessive doses, producing various forms of arrhythmia, block and tachycardia. These disturbances may be quickly detected in hospitals by the electrocardiograph, but in family practice the physician should be cautious upon the appearance of any new arrhythmia. Quinidine sulphate, apparently, has also this power to poison the heart and must be used with care. I have seen cases in consultation where quinidine was being given as freely and thoughtlessly as digitalis.

In closing let me say to many of you who are in the practice of general medicine that since disturbances of the heart make up a considerable portion of your work, it adds greatly to your satisfaction and the benefit of your patients to understand the mechanism of the normal and abnormal heart. Many disturbances of the heart come not because of pathological changes in that organ but from entirely extraneous sources. The days of treatment with digitalis, or some allied drug, of any suspected heart defect are giving way to an intelligent understanding of physiological and pathological cardiac mechanisms with treatment directed largely to improperly functioning diseased heart and blood vessel tissues. Heart disease can be very well diagnosed and treated by the trained general practitioner. The specialist must continue to work out the meaning of the various mechanisms and to elucidate them to his students and colleagues, to indicate proper and new forms of treatment and to consult in cases of intricate cardiac and circulatory abnormalities.

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CYSTINURIA COMPLICATED BY CYSTINE CALCULI WITH A CASE REPORT*

BY JAMES B. HICKS, M.D.†

CYSTINE (C6 H12 N2 S2 O4), as its name implies (Kystis, bladder) was originally found in bladder stones. It is an oxidation product of cysteine (C3 H7 S O2), an amino acid derived from protein metabolism. The latter is readily oxidized, forming cystine and water. Both compounds are found in proteins, while only cystine occurs in urine. Very small amounts occur normally in the urine. Such individuals as have cystine in the urine in abnormal quantities are said to have cystinuria.

This abnormality is probably due to an unexplained error in protein metabolism. According to Brand, Harris and Biloon[1], in cystinuria a cystine compound is excreted which decomposes in the urine with the liberation of free cystine. In the case on which their studies were made, it was found that in the freshly voided specimen, after the patient had been taking sodium bicarbonate, there were no cystine crystals present. After the specimen was allowed to stand for some hours crystals formed. These authors also showed that the cystinuric patient oxidizes free cystine to the same extent as the normal and excretes it as an inorganic sulphate, because when a cystinuric is fed cystine crystals, the neutral sulphur in the urine remains fairly constant and little, if any, additional cystine appears in the urine.

Cystinuria is an hereditary disorder and has been traced through three generations. Cystine crystals are only sparingly soluble and are, therefore, in the process of excretion, apt to form urinary calculi. Many individuals have cystinuria for many years or a lifetime and never develop calculi, while others do, probably because of urinary stasis and infection. It is when calculi are present that cystinuria becomes of clinical importance.

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Small cystine stones are only slightly, or not at all, opaque to the x-ray, and, consequently, in this condition no information is apt to be obtained by this means. Whenever a patient has pain suggestive of renal colic, with the presence of cystinuria, cystine stones should be suspected and sought by other studies regardless of a negative x-ray. The large hexagonal cystine crystals (resembling mother-of-pearl) are typically present in the urinary sediment of cystinuria. When these are found in the above situation, the diagnosis is fairly certain. Cystoscopic study adds evidence which usually clinches the diagnosis. The actual stones can, of course, be examined chemically. The authors quoted above suggest cyanide nitro-prusside reaction as a test for cystinuria. This is carried out as follows: To 3-5 c.c. of urine add 2 c.c. of a 5% NaCN. Allow to stand for 5-10 minutes. Then add a few drops of 5% solution of sodium nitroprusside. Normal urines develop only a faint color, while cystinuric urines give a rather stable magenta color, which is characteristic for the SH group.

No treatment is indicated for cystinuria unless calculi manifest themselves. Osler[2] states that two things may be of value: the protein in the diet may be reduced and large doses of sodium bicarbonate (6-10 grams daily) by mouth may be given. The sodium bicarbonate increases the solubility of cystine. Surgery is employed in the removal of obstructing calculi, but should only be undertaken with the realization of the likelihood of the return of the calculi.

During the last year a patient with cystinuria and cystine renal calculi was treated in the Lahey Clinic. Inasmuch as this patient was handled successfully with the cystoscope and since this is so unusual a condition, we think a report of the case is worth while.

CASE REPORT

The patient, a white man aged 26, was first seen March 13, 1929 with a complaint of pain in the left side of the abdomen. His past health had been generally good. Except for numerous attacks of tonsillitis, for which his tonsils and adenoids were removed, and an appendectomy, there had been no illnesses of consequence. During the last five years previous to coming to the Clinic, he had had about thirty attacks of severe colicky pain in the left kidney region, radiating along the flank to the left inguinal region. The attacks lasted from a few hours to five days. There was nausea and vomiting associated with the attacks, also some frequency of urination. He came to the hospital with a typical attack, which had begun three days previously. The urine had been grossly bloody on two occasions during the last two days. He had recovered about 27 stones from the urine following attacks of pain. These had been analyzed and found to be cystine. The patient stated that the stones on being passed were snowy white and became brown after a few days, resembling lumps of grape sugar.

Physical Examination—The patient was a well developed and nourished white man, aged 26, who was having colicky pain in the left side of the abdomen at the time of examination. His temperature was normal. There was moderate distention of the abdomen and marked tenderness over the left kidney, both anteriorly and posteriorly. The physical examination was otherwise essentially negative.

The urine showed a few red and white blood cells. Some of the white cells were in small clumps. Examination of a stained specimen of the bladder urine sediment showed rod-like gram-negative bacilli. The plain x-ray showed no evidence of calculi anywhere in the urinary tract. The kidneys were not clearly outlined because of large quantities of gas in the intestines. On the day following admission to the hospital, a cystoscopic examination was made under gas anesthesia. A F24 Brown-Buerger cystoscope was introduced without difficulty. The bladder was of good tone and good capacity. The urine evacuated was slightly cloudy. Lying on the floor of the bladder were many snowy white crystals. The mucosa was everywhere essentially normal. There was no evidence of bladder neck obstruction. No stones and no tumors were seen. Both ureteral orifices were normal in appearance. At short intervals white crystals were ejected from the right ureter with otherwise clear jets of urine. Nothing was emitted from the left. Indigo-carmine given intravenously appeared on the right side in good concentration in four minutes. None was seen on the left during the whole operative procedure, which lasted about forty-five minutes. All catheters introduced into the left orifice met with impassable obstruction at a distance of 2 cm. from the bladder. The orifice was enlarged with Bumpus scissors and dilated with alligator forceps. Attempts were made to introduce a Howard-Welland stone dislodger. This also met obstruction at the same level as the catheters. During its use, however, a small fragment of stone was dislodged and brought down into the bladder.

A cystoscopic manipulation was again done four days later, when the orifice was still further enlarged by fulguration and the calculus brought down within sight, but could not be completely delivered into the bladder.

The patient was discharged the following day. He did not have any further pain and recovered the calculus in the voided urine a few days later.

A cystoscopy was done and pyelograms were made on April 15, 1929 (see Fig. 1). At that time there was injection and edema about the left ureteral orifice, which was large and patulous. There were also tags of mucopurulent material adhering to the mucosa about it. There was a moderate amount of pus in the bladder urine. Indigo-carmine given intravenously appeared at the right orifice in 3½ minutes and at the left in 4½. Both ureters were easily catheterized with No. 6 catheters. Following the examination three pelvic lavages of the left kidney with ½% silver nitrate solution at weekly intervals, with the patient on forced fluids, were given. At the end of the course of treatment the inflammatory changes about the orifice had disappeared and there was no pus in the urine. On completion of the cystoscopic treatment, he was urged to drink at least ten glasses of water daily. No limitations were put on diet except that he was instructed to avoid excesses in any foods and to partake only very moderately of alcoholic beverages. No internal medication was given.

There has been no further attack of pain and the urine has remained pus free.

In view of the condition being hereditary in nature, the urines from the members of the patient's immediate family were examined. From these ex-

aminations the father and two brothers were found to have cystinuria, while no evidence of it was found in the specimens from the mother and a sister. None except the patient gave a history of ever having any symptoms suggesting the presence of calculi formation.

The significant points in this patient's history and examination which made us suspect cystine stones were the repeated attacks of renal colic over several years, with negative x-rays and the presence of pearly white crystals in the urine sediment.

It is worthy of note that in this condition an actual calculus may form and conservative treatment should be used if possible since further calculi are so prone to occur.

In reviewing the treatment, it is natural to wonder why we did not employ diet and alkalinization of the urine. We felt that, inasmuch as other members of his family had cystinuria with no evidence of having calculi, and also in view of the fact that his attacks were always left-sided, he must have stasis or infection localized to that side which, if corrected, would rid him of his trouble. There was no evidence of stasis by pyelographic study, but there was infection. We chose to use cystoscopic treatment as the surest way of eliminating this.

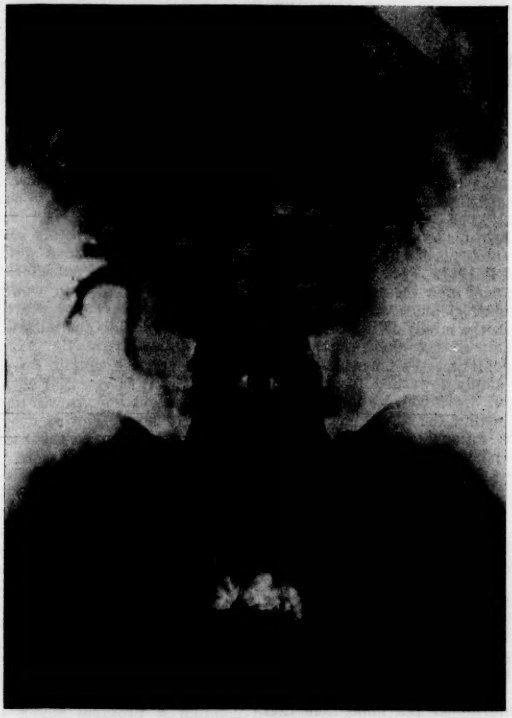

FIGURE 1. Cystine renal calculi.

SUMMARY

Cystinuria is an hereditary disorder due to some unexplained error in protein metabolism. It is fairly common, while cystine calculi are rare. When calculi are present, the condition becomes of clinical significance. Some methods of diagnosis are presented. Dietary measures and alkalinization of the urine, with sodium bicarbonate, are recommended in the literature. The correction of urinary stasis and elimination of infection, as in other forms of urinary calculi, seem to be the valuable treatment. A case of a young man with cystinuria and left renal or ureteral calculi, who has been treated successfully cystoscopically, is reported.

NOTE

After the above report was prepared for publication, the patient again reported to the Clinic complaining of slight frequency and burning on urination. The urine was sparkling clear, but a microscopic examination of the sediment showed a rare white blood cell. There was also an occasional pus cell in the prostatic secretion. On cystoscopic study a vesical calculus about the size of a pigeon's egg was found, along with a narrowing of the bladder neck. The kidneys functioned normally. The calculus was removed with a lithotriptoscope, and the bladder neck dilated at intervals since then. At present he is again symptom-free and there is no pus in the urine.

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THE FINDINGS OF THE COMMITTEE ON GROWTH AND DEVELOPMENT OF THE WHITE HOUSE CONFERENCE ON CHILD HEALTH AND PROTECTION*

BY HAROLD C. STUART, M.D.†

THE findings of the Committee on Growth and Development were very concisely presented by the Chairman and other members at the February meeting of the Conference. These summaries have been made available in printed form in the special issue of the *United States Daily* dated April 6, 1931. They gave emphasis to the newer points of view regarding the problems of growth and development which had come into prominence in the minds of the Committee members as a result of their deliberations.

There is no doubt that the members of this Committee were amply rewarded for the time spent in study and discussion, by having their opinions checked against the known facts and by obtaining a more rational concept of relative values in matters pertaining to child health. If I understand the purpose of this Massachusetts White House Conference Institute, it is to pass on to the people of this State who are actively participating in work for children some of the advantages which accrued to the Committee members themselves. For this purpose, I shall quote frequently from the addresses referred to and elaborate, in so far as time permits, from the wealth of useful information contained in the final reports of the Committee. These reports are now in the hands of the printers and should soon be available for more detailed study. I fear they will be a disappointment to many people who had entertained the hope that they would solve their problems and simplify their procedures. On the contrary, they make the undertaking of sympathetic observation, protection, and guidance of mental and physical growth a far more difficult task than it is commonly believed to be. They discourage the too ready acceptance of evidence derived from our present norms of measurement, and the standardization of routines and diets for children of different ages which disregard individual differences. The reports are, however, no cause for discouragement in regard to our ultimate objective. Ample evidence is presented to justify the belief that many factors which now interfere with normal growth and development can be eliminated by intelligent effort and that we are going rapidly forward in acquiring scientific information which can be practically applied to bring about better mental and physical health in growing children.

To understand many of the conclusions of the Committee, it is essential to grasp the fundamental concept that growth and development is an orderly sequence of events governed by natural laws common to the race but subject to marked individual variations. These variations are in large measure dependent upon heredity and other chance factors outside of human control. Although these deviations are, at times, so conspicuous as to mask the underlying orderliness and predictability of growth, yet the lawfulness of growth is traceable through all its processes. We are still in complete ignorance as to the factors which determine individual variations, and, even if many of them are potentially under our control, we are not yet in a position to say which combination of variables is the most desirable from the point of view of health. It is of the utmost importance, therefore, that, in planning a program which contemplates the conscious modification of factors effecting growth and development, we be quite certain first that our objectives *are* desirable, and secondly that there is good reason to believe that they are obtainable.

The individual variations in the normal growth processes are so extreme and diverse that the commonly used phrase "the normal child" eludes definition. It is impossible to sharply differentiate those findings which are presumptive evidence of abnormality in an important health sense from those that are merely extremes of the normal distribution curve. This makes it

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impossible to formulate objective standards which can be applied to determine individual normality.

Much can be accomplished in protecting growth and development in children through the usual public health activities applied to groups, and through educational methods directed toward both parents and children. But if protection is to be as effective as our knowledge permits, health service must be available to the individual child at all periods of the growth process. The newborn infant, during the first few weeks of adjustment to independent existence, is a highly unstable organism. The changes resulting from growth and development are very rapid at first, but become progressively more gradual over the first ten to thirteen years until the organism is relatively stabilized. Shortly before puberty, it again undergoes rapid alterations in structure and function, while noticeably increasing in bodily dimensions. Following this there is a final, but gradual, movement toward the steady state of maturity. It is curious that the two periods of greatest instability, the so-called neonatal, or newborn, and the adolescent, have received relatively little attention in health projects. Although our knowledge of the newborn is inadequate, we know a great deal more concerning the importance of early examination, of effective isolation, of establishment of special environment for the feeble and premature, and in general of the value of intelligent health supervision at this age than is given expression in general practice.

An analysis of the possibilities for protection of children during the growth period recorded or implied in the committee reports suggests the following grouping of subjects:

I. Health examinations—that is appraisement of the physical and mental condition of individual children to determine their fitness and special needs.

II. Provision of the nutritional requirements for normal growth and development.

III. Protection from disease or other environmental handicap detrimental to satisfactory growth and development.

IV. Guidance and training in habits of thought and action which are favorable to growth and facilitate well-balanced development.

Let us first consider a few of the points emphasized by the subcommittee on physical status relating to health examinations.

I. Appraisement of the Child

In making a health appraisal, we aim to determine the extent of growth and the maturity of development which the child has attained. We wish to judge his fitness for everyday life from physical, mental, and social points of view and we should be in a position to make some prognosis as to his probable physical and mental attainment at maturity. The examiner must remember that each child has his own potentialities determined primarily by his inherited physical and mental equipment, but almost inevitably modified by the environment in which he has lived. The goal of any complete appraisement is to determine how closely the child is approaching the full realization of these possibilities, and what interfering factors could be removed. It requires training, experience, and vigilance to detect those deviations from the so-called normal which constitute actual or potential handicap.

It is clear that the foundation upon which supervision of the growth and development of any child must be built is a thorough understanding of the child himself with particular reference to his personal limitations as well as his outstanding capacities. Comprehensive appraisement must include a study of the child's family history, the suitability of his environment, and his own adjustment to this environment. It calls for knowledge regarding the occurrence of congenital defects and disease in his past history, and the detection of disease processes or abnormalities still present. This information will be more accurate if collected from time to time throughout childhood as a personal health record. A complete and thoroughgoing physical examination carried out by a physician experienced in dealing with both normal and sick children is necessary to determine adequately a child's present state of health. In the last analysis the interpretation of physical findings is based upon comparison with other children of like age and circumstance. It is important that our standard of comparison be one of positive health and not merely one of relative freedom from disease.

The logic of comparing a measured aspect of one individual to a series of similar measures for other individuals like him in certain other ways is unassailable; but our present norms are based upon large heterogeneous averages and not on groups representing *desirable* combinations of heritage, home influences, and health history. All measurements need thorough evaluation before they may be made the basis of judgments about individuals; and one would do better to depend upon the subjective evaluations of experienced observers than to carelessly apply the more objective findings of measurements. Many of the problems inherent in the interpretation of findings at a single examination are solved by repeated serial examinations which allow consideration of progress.

Although time will not permit our dealing with other aspects of appraisement specifically, we will consider some of the problems associated

with the determination of nutritional adequacy, as these have wide application and popular interest.

METHODS OF JUDGING THE ADEQUACY OF NUTRITION

How may we select from a group of children those whose nutrition is inadequate and who require special attention as to diet and régime? The simple procedure of taking height and weight and relating these to tables of averages for children of similar age and sex is inadequate. Differences thus found are largely due to differences in build. We might overcome this difficulty to some extent by taking certain other measurements, such as breadth of chest and hips, and compare the individual under consideration with groups like him in these respects also; but this introduces many complications. No such tables are at present available, but such comparisons as have been made show that differences not attributable to bony development are small in normal children.

We may gain more useful information by taking serial measurements on the same child and comparing his present status with that found at some previous period, thus paying attention to the progress made. Such measurements are of much greater significance and are very useful for large items such as weight and height. For many measurements, however, these increments are so small as to be masked by the technical errors involved.

In the last analysis measurements all deal with quantitative changes, and yet the most important factors to be judged in regard to the adequacy of nutrition are the qualitative aspects of the tissues being judged. Everyone knows that the biggest, fattest baby may really be the poorest specimen from a nutritional standpoint, the soft, flabby tissues and the poorly formed bones being due to inadequate proteins, salts, or vitamins. We must, therefore, in any appraisal give consideration to quality of tissues. The elasticity of the subcutaneous tissues, the tone of the muscles, the size and shape of the bones, the eruption pattern and quality of the teeth, and even the appearance of eyes, mucous membranes, and hair are all significant. We have in the x-ray a valuable method of judging the efficacy of nutrition in producing good quality of bone growth as well as in judging the normalcy of bony development. Although not available at present for extensive use in health appraisals, it is to be hoped that this approach will be utilized more and more as time passes.

Briefly then, we may continue to use height and weight and other measurements in conjunction with physical examinations in our screening of large groups of children, but the findings on such screening should only be interpreted as indicators, never as criteria of disease or malnutrition, and should be supplemented by more extensive individual study before conclusions as to diagnosis and treatment are arrived at.

The measures of muscle and subcutaneous tissue being developed by the American Child Health Association may prove to be very useful expressions of the adequacy of the nutritional processes; they give promise of being more significant than weight in relation to height and age. The Committee, however, is not prepared to recommend the routine adoption of any measurements as *criteria* of nutrition, growth, or health.

Let us now consider a few points in the report of the subcommittee dealing with nutrition:

II. Nutrition

The selection of an appropriate diet is merely one of a variety of prophylactic medical measures used to insure protection against the encroachment of disease and other limitations of growth and development. The reasons for giving attention to diet are first, and most important, because science has demonstrated that a number of different dietary deficiencies are related to specific forms of disease or interference with normal growth. There is also a second important consideration—namely that food may be the vehicle for introduction of various pathogenic organisms (or less frequently other insidious poisons) into the system; and lastly that mechanical injuries may result from dietary indiscretion.

In connection with the prevention of dietary deficiencies, it is important to have a clear understanding of the significance of the "law of the minimum" in the domain of nutrition. This law maintains that there is a minimum quantity of each of the essential food ingredients which must be provided in any given diet if that diet is to prove adequate. No matter how abundant and well planned a diet may be in other respects, it will inevitably lead to serious consequences if it contains less than is required of any one of the food essentials. There are wide age and individual differences in the minimum amounts required, and we are far from being able to state in a given case what quantities of each constitute an irreducible minimum, so we must provide liberally. This is emphasized by the newer concept that gastro-intestinal defects cause faulty absorption of dietary essentials. Furthermore, we are not yet in a position to give a complete list of all the essential elements. Dr. Mendel, however, concisely stated in his summary that there are thirty-five relatively simple substances which comprise the adequate diet in so far as we understand it today. Eighteen of these are the end products of protein digestion. One is sugar, glucose; ten are mineral elements; and six are vitamins. So far as we know, any food supply which provides all of these thirty-five principles in their proper amounts will allow normal growth in the young.

There have been at different periods various degrees of stress laid upon different individual elements in this list. As newer elements have been discovered special stress has been laid upon them, and we are now in the stage of emphasizing the importance of vitamins, but it should be kept clearly in mind that all of the thirty-five substances are essential and none of them can be safely neglected.

In addition to the provision of the minimum requirements of each of the essentials of diet, it is important to assure an adequate number of calories to meet the individual's energy requirements. We need better predictive standards for energy requirements than those in common use. Evidence is presented which indicates that for adolescence, at least, the requirement for energy substances is greater than has previously been believed, and the modern trend of scientific research suggests that we are not justified in skimping our youth in matters of diet. A lengthy consideration of the probable requirements by age, etc., is contained in the final report.

Little need be said to this audience regarding the importance of protecting our children from the taking of contaminated foods. The progress made during the last generation in the reduction of infant mortality from gastro-intestinal disorders is an indication of the importance of pure food in early life. This progress has been in large measure due to the general substitution of clean for contaminated milk. Speaking of milk suggests the question which is frequently raised as to why we give such prominence to it in education pertaining to diet, and I might quote from Professor Rose's answer to this question. "Milk owes its importance in the diet to the excellent quality of its proteins and their supplementary value to the cereal proteins, to the completeness of its assortment of mineral elements and the excellent proportions in which they occur, to the high content of calcium which makes milk almost (but not absolutely) indispensable for ideal storage of this element during growth, to the presence of vitamins A, B, and G in quantities which make generous use of milk day by day a practical guarantee against deficiency of any of the three, and to the presence of an appreciable amount of vitamin D in association with a calcium phosphorous ratio very valuable for the formation of bones and teeth." We must not forget, however, that milk has certain shortcomings. In the first place it is an excellent medium for the dissemination of pathogenic organisms, unless subjected to the most painstaking supervision. Its content of each of the vitamins is quite variable, depending upon the cow's rations. We know that breast fed infants develop rickets during the late winter months because their mothers do not receive enough vitamin D either during pregnancy or lactation. All milk is a relatively poor source of vitamin C, of iodine, and of iron. There is little doubt that a large amount of milk in the diet interferes with appetite in some children and with the taking of adequate amounts of other food substances. We also know that an excessive amount of milk may lead to digestive difficulties.

In our enthusiasm for making certain that every child receives his full requirements of each and every food substance, and in our efforts to provide a wide margin of safety, not knowing exactly what the minimums are, we cannot overlook the possibility of unwittingly doing harm in a variety of ways. There is definite evidence accumulating that excessive intake of certain vitamins may be harmful and this suggests caution in the indiscriminate use by the public of our new highly potent artificial vitamin substances (such as irradiated ergosterol) as a food rather than as a drug. There is the very pertinent question as to what is optimal growth. Even though we may be able to produce larger babies by increasing their intake of certain food substances, are we necessarily making better babies of them? Again, what effect does too great insistence upon details and too much concern about the feeding habits of our children have upon their psychological reactions to the taking of food? The Committee report deals at some length with this element in the situation because there is ample evidence that large numbers of children develop undesirable habits of eating, as a result of the attention to diet which is constantly in evidence. The report states that there is no ground for the belief that the conventional American diet is the only adequate one; on the contrary, most of the native diets of the different races are far superior. An adequate diet may be secured in a variety of ways. Wise dietary advice consists in determining the deficiencies in a particular diet and in making only such changes as are necessary to supplement or correct these deficiencies, rather than trying to make all diets conform to our own particular notions as to what is ideal.

Under our third heading, protection from disease or other environmental handicaps, we may properly consider three topics, (1) infectious disease, (2) atmospheric conditions, and (3) other environmental factors.

III. (1) *Infectious Disease*

The effects of infectious disease on growth and development are very hard to measure because of our inability to determine exactly what any given individual would have been like, if a certain infection had not occurred. There is considerable evidence that most infections temporarily interfere with growth and that this interference is most evident in the processes which

are going on most rapidly at the time of the disease. The effects of infection are recognizable, for example, in the delay in epiphyseal maturation shown by the x-ray. This retardation in growth is usually quite temporary and limited to the period of active disease. There is a definite tendency for growth to be accelerated during the period of convalescence in order to compensate for the preceding delay. At times this convalescent acceleration exceeds the retardation due to the disease, so that its net effect is a stage of growth beyond that which might normally have been expected. Todd has found evidence in x-rays that this occurs in bones following attacks of measles. Although our evidence is not really adequate, we may conclude with reasonable assurance that most acute infectious diseases do not materially interfere with the ultimate attainment of potential growth, except in so far as they produce permanent damage to localized tissues. The more long-standing infectious diseases, particularly when they are active during the rapid periods of growth, are more likely to leave the body stunted; but even here there is a surprising lack of evidence that this stunting lasts to maturity. In other words, the capacity for compensating for early retardation is very great and for practical purposes we may think of the permanent effects of the infectious diseases as defects due to localized destruction of cells or tissues. The temporary effects are, however, often marked and the compensation taking place during convalescence adds very special requirements for food and rest. The period of convalescence in children is a far more important one than is usually appreciated and calls for closer supervision to prevent undue stresses and to assure adequate nutrition.

We are particularly anxious to prevent infectious diseases during the early years of rapid growth primarily because we know that the case fatality from most of them is far higher at this period, and secondly because local destruction of cells and tissues may interrupt or permanently arrest certain growth processes. For example, our primary reason for wishing to prevent whooping cough under one year of age is not because of what whooping cough does to the growth processes, but because of its extremely high case fatality under one year compared with its relatively insignificant fatality rates in later childhood.

(2) *Atmospheric Conditions*

Our Committee dealt at some length with the evidence relating to the effects of atmospheric conditions upon growth and development. That relating to sunlight is voluminous. We have evidence that sunlight acts upon the ergosterol normally present in the skin, and synthetically produces vitamin D. We know that sunlight has a very definite effect upon calcium metabolism and is a specific in the treatment and prevention of rickets and tetany. The evidence at hand does not justify many of the other contentions in regard to its usefulness. It has not, for example, proved to be a general measure of usefulness in increasing resistance to infection; nor has it been proved to affect beneficially the development of normal blood constituents, or to noticeably accelerate the rate of growth. One of our reviewers states that "there is no justification for our being light faddists either as individuals or as practitioners. The way of hygiene is the way of moderation not only with respect to light but with respect to most of our environment, and in maintenance of balance between the items." The report dealing with other atmospheric conditions had of necessity to deal largely with morbidity and mortality rates and these could only be related to growth and development by inference. There is a well-marked seasonal variation in the incidence of most of the common diseases of childhood. There are indications that children grow in weight in the summer and autumn more than in the winter and spring; but that they grow in height to a greater extent at the inverse seasons. How much of this is directly attributable to atmospheric conditions and how much to other seasonal factors, such as the incidence of infection, is debatable. No standards of ideal atmospheric conditions for optimum growth and development can be set up. For persons in the northern and eastern parts of this country, it would appear that an average outdoor temperature of about 63° F. and reasonably high relative humidity are on the whole desirable and that open air under favorable conditions is preferable to indoor air. Indoor temperature of 68° to 70° with relative humidity of 60° to 65° and air in motion have seemed to be associated with the best health results.

(3) *Other Factors*

It would seem obvious that a child cannot grow and develop normally if environmental conditions are such that he cannot obtain an adequate amount of sleep, of rest, and of exercise. Our knowledge in regard to the sleep requirements of infants and children at various ages is largely based upon observation of the sleeping habits of series of supposedly normal children living under conditions favorable to the taking of as much sleep as they desire. We know very little about the direct effects of reducing the time allotted to sleep at the various ages. A great deal has been written on the subject of chronic fatigue, its causes and its effects; but signs and symptoms of chronic fatigue are not easily distinguishable from the signs and symptoms of latent disease and it is most difficult to measure directly the effect of

fatigue. That chronic fatigue is an important problem in both pre-school and school children deserves wider recognition for it is one of the things we can definitely improve by wiser planning of routines for individual children. Chronic fatigue and infection are often associated and form a vicious circle which can at times be broken by a rest routine. We should attempt to provide conditions suitable for the taking of as much sleep and rest as nature calls for in the absence of undesirable stimuli, and we should offer full opportunity for taking natural forms of physical activity upon which normal growth and development is undoubtedly dependent. Our object here, as in other instances of health supervision, should be to prevent or remove unnatural barriers against usual or desirable habits.

This leads us to the subject of habit training.

IV. Mental Growth

First, a word as to our knowledge regarding mental growth—The study of mental growth must be largely through observation of behavior. Behavior patterns have been found to be very consistent and lawful in relation to age and other associated factors for use as diagnostic indices. Considerable progress has already been made in the formulation of these patterns. By this method we may hope to determine the tempo, trend, and configuration of individual mental growth with as much satisfaction as we can determine these characteristics of physical growth by direct measurement. These methods are, however, still in a rudimentary stage and normal criteria must be used with due clinical caution and supplemented with clinical judgment. Here again the accuracy of determinations will depend largely upon the results of periodic re-examinations and consideration of growth increments and characteristics rather than on momentary status. The study of behavior is of importance to preventive medicine and particularly to pediatries because behavior is a sensitive index of physiological and dynamic status and is very definitely influenced by conditions of physical health and disease. The report deals at some length with the present techniques for the study of intelligence and behavior, and for the measurement of motor skills and language at the various ages. The conclusion is reached that we are still handicapped by our lack of adequate techniques and standards with which to measure practically every aspect of mental life, with the possible exception of intelligence, and that oftentimes we are still confused as to what we are really trying to measure. We have gone far enough, however, with the study of mental growth to be quite certain that behavior is orderly and predictable, and that it has pattern and is measurable. We should be extremely hopeful as to the practical usefulness of further studies in this field.

Habit Formation and Education

Our Committee simply attempted to review the part played by habit formation and training in determining the course and ultimate attainments of mental development. It gave no consideration to educational methods dealt with by other committees.

If one confines his attention to intellectual capacity, it must be admitted that training and environment are of much less importance than inborn capacity. Education and practice will never make a feebleminded child normal or create genius from mediocrity. But the report states that "the slowly accumulating evidence indicates that the mental development of the child may be encouraged and accelerated to a very material degree by the application of the most effective methods of instruction." It even goes on to suggest that by constantly improving the methods of education we may hope to raise materially the level of intellectual performance of both children and adults. Our interest in mental development is, however, far broader than intellect and is actively concerned with the child's behavior and his ability to make the necessary adjustments for successful and happy living. We know that behavior is very largely determined by training and early experiences, and that environmental influences are of the utmost importance in determining the future course of conduct.

All through the report of our Committee the unity of the individual and the interdependence of physical and mental characteristics are stressed. It is unreasonable to suppose that growth and development may be sympathetically observed and fruitfully protected by attention only to physical factors on the one hand or to mental factors on the other; and the report urges attention on the part of physicians supervising the physical health of children to the problems of mental development, habit formation, and training. As one of our Committee members expressed it "the physician who is to be successful in conducting periodic health examinations . . . will be the one who learns to recognize and deal with problems of training and behavior as well as those of weight and height and physical defects, who is familiar with the various stages of mental development, who has learned to judge when expert psychiatric or psychological advice is needed and who appreciates the influence of the social and economic environment upon both physical and mental development."

CONCLUSION

We may conclude from the studies of our Committee that the growth and development of individual children is modified by many factors that are under our control. Efforts to pre-

vent those factors which interfere with normal growth should be encouraged, and to be most effective should apply to all periods of the growth cycle from conception to full maturity. A redirection of emphasis is essential in the light of our newer knowledge; but the present approaches through health examinations, dietary and environmental supervision, prevention of disease, and education are worthy of further development.

TREATMENT OF PAROXYSMAL TACHYCARDIA*

BY JAMES C. HEALY, M.D.†

PAROXYSMAL tachycardia is a well recognized and, in a large percentage of cases, a functional cardiac disturbance. A fair number of patients with this affection are perfectly well at the time of consultation and the diagnosis is made on the history of sudden onset of palpitation and the absence of heart or thyroid disease as determined by x-ray, electrocardiograms and basal metabolism rate. Treatment at such times is unsatisfactory and generally limited to instruction of the patient in the art of stimulating his own vagus nerves by pressure.

If seen during an attack, immediate relief is urgent and two forms of therapy are indicated, mechanical or medicinal, or sometimes both. It is noteworthy, however, that any mechanical attempt to stimulate the vagus often fails, and that the same method is liable to be valueless in a second attack. The operator has the choice of pressure in the supraorbital region, on the eyeballs, sinus caroticus or abdomen, or of having the patient sip cold water and attempt expiration at the same time. Czermak's manoeuvre, massage from above downwards along the medial border of the sternomastoid muscle with deeper pressure as the jugular notch is approached, may be successful.

The second, or medicinal, form of therapy involves a variety of drugs, some being empirical, others having fairly well understood modes of action. Digitalis and quinidine sulphate, by their action on the normal and abnormal conduction system, seem to be the most indicated and popular drugs. Successful use of the former, not only in acute attacks but also to prevent recurrences, has been reported by several authors. Levine and Blottner[1] in particular report on its effectiveness and advise using large enough doses to ensure its success. Such large doses, aside from the subjective symptoms of intolerance to the drug, may lead to complications, especially the onset of a ventricular tachycardia during digitalis therapy. Eakin[2] also endorses the use of the drug, but warns of the possibility of a ventricular tachycardia arising during treatment. In such an event digitalis is contraindicated and is, in fact, the etiological factor. Howard[3] urges electrocardiographic examination in all cases where rapid heart action ensues during such treatment, to ascertain the exact diagnosis, lest the condition be made worse by forcing more digitalis.

Singer and Winterberg[4] report six cases of paroxysmal tachycardia in the acute stage relieved by intravenous administration of quinidine sulphate, the average dose being .5 grams. Symptoms were relieved and the pulse normal ten minutes after injection. Scott[5] and Gilbert[6], reporting similar cases, found that .4 grams quinidine sulphate by mouth checked every attack in a few minutes. After the acute tachycardia had subsided, the patients were put on a course of treatment, receiving at first .6 grams, later .2 grams a day, with no recurrence during six months. The drug was then omitted and an attack ensued. The same treatment was repeated with no recurrences. Sprague and White[7] attest the value of quinidine sulphate in about one-half of the cases of paroxysmal tachycardia, the rest being unimproved.

Stepp[8], in 1925, described the use of cholin and its derivatives intravenously. It had been noted that in experimental dogs after intravenous injection the heart slowed appreciably. In one patient with paroxysmal tachycardia an intramuscular injection of 1 c.c. of a 20% cholin derivative solution caused a fall in heart rate from 240 to 84 in five minutes. The only disadvantage is the extreme substernal discomfort which the patient experiences. In an emergency the drug may be used intravenously, but it should be well diluted with physiological salt solution and injected very slowly.

Kaufmann[9] and de Meyer[10] reported the good results obtained with strophanthus used in conjunction with 1.5 to 3. milligrams of physostigmine and administered subcutaneously. v. Hoesslin[11], working along the same lines, employed pilocarpin in 10 to 20 milligram doses hypodermically. He stressed the importance of sedatives, narcotics and even anesthetics in the treatment and drew attention to the value of psychic influences.

Intravenous injection of 1 c.c. epinephrine solution 1-1000 has been successful in the hands of Schuster[12], after subcutaneous administration of the same drug had failed. Desiccated parathyroid gland in 1/10 gr. doses three times a day has been used with success by Dukes[13] as a rem-

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edy in the attack and as a preventive in the interim.

Recently (1931), Wolffe and Bellet[14] have reported the successful use of calcium gluconate in 3 cases of paroxysmal tachycardia. The drug is administered intravenously, at a slow rate, and in their experience gives prompt relief. The mode of action is not understood clearly, but they indorse the method in those cases where other measures have failed.

The following case reports of patients with paroxysmal tachycardia are presented, together with their treatment. In studying these cases the patients' diets were carefully scrutinized and all were found to be low in fat and high in carbohydrate. It was also ascertained that no patient had been taking any alkaline drug.

CASE No. 1.

C. R., a married woman of 68 years, was seen about one hour after the onset of attack. The history as given by her husband revealed the fact that ever since their marriage, 50 years before, she had been having frequent attacks of palpitation, fainting and shortness of breath, lasting from a few minutes to several hours. For the past 2 years the attacks had become so frequent and exhausting that the patient refused to leave her home and had become a semi-invalid. For the previous 2 weeks the attacks had recurred about every 2 days, lasted 6-8 hours, and were preceded by an olfactory aura. She had had a variety of medical and osteopathic treatments and on some one's advice had been living on a fat-free diet for 2 months with a rapid loss of weight and a marked increase in the severity of the attacks. For 6 months she had been taking 2 grains of digitalis a day.

Present History. Unimportant.

Marital History. Married 50 years. Two uneventful deliveries. No miscarriages.

Catamenia ceased at 46 years.

Physical Examination. The patient was in marked distress, unable to talk, slightly cyanotic, and perspiring freely. Her skin was lax and the pulse too rapid to count at the radial artery. There was moderate thickening of the brachial and radial arteries. Heart sounds at the apex were regular and of good quality; rate 180. Vagus stimulation by ocular pressure relieved the attack, but the pulse regained its rapid rate after an intermission of 15 minutes. Pressure on the carotid sinus, on the abdomen, and sips of cold water each relieved the condition for a few minutes. The patient was given morphine and the tachycardia disappeared suddenly 8 hours after onset.

An electrocardiogram, taken the following day, proved to be normal.

Urine, taken 4 hours after recovery, was found to be strongly alkaline, 100 c.c. requiring 24.3 c.c. N/10 hydrochloric acid for neutralization. Other tests, including the phenolsulphonphthalein and the concentration test, failed to demonstrate renal pathology.

Morning urine specimen on the second day was less alkaline, 100 c.c. requiring 12.6 c.c. N/10 hydrochloric acid for neutralization. The third morning the reaction was almost neutral, .35 c.c. N/10 hydrochloric acid being sufficient to decolorize the phenolphthalein in 100 c.c. urine. Fortunately a urine specimen was obtained during an attack that afternoon, which was acid to litmus and required 18.15 c.c. N/10 sodium hydroxide to neutralize it. Two hours later it was strongly alkaline again, requiring 20.8 c.c. N/10 hydrochloric acid for neutralization.

Assuming that the case of paroxysmal tachycardia, in this instance at least, might be due to an impaired acid-base equilibrium with sudden loss of acidic salts and an elevated alkaline concentration in the tissues and plasma, the following treatment was instituted at the onset of an attack 2 days later. Forty minims of dilute hydrochloric acid and 4 ounces of whiskey were administered by mouth. Various forms of vagal stimulation were attempted with momentary relief. At the end of an hour the pulse dropped to normal.

The patient was then instructed to take 5 grs. of acetyl-salicylic acid 4 times a day and a high-fat diet prescribed. Daily urine tests were done for three weeks, the average acidic coefficient being 26.4 c.c. N/10 sodium hydroxide per 100 c.c. urine. Six weeks after the above treatment the patient was again seen in a mild attack of paroxysmal tachycardia, which was quickly relieved by a dram of dilute hydrochloric acid and ocular pressure. A specimen of urine was collected immediately after the attack and 100 c.c. required 6 c.c. N/10 sodium hydroxide for neutralization. Specimens of urine taken at irregular intervals in the past five months have all been of normal acidity. The patient has gained weight, is symptom-free and has been entirely free of any alterations in heart rate or rhythm.

CASE No. 2.

F. H. C., a young married woman of 25, was seen in a typical attack of paroxysmal tachycardia in December, 1930. Her history revealed the fact that ever since puberty at the age of 16, she had had attacks of severe palpitation, breathlessness and exhaustion on the first day of each menstrual period. These attacks lasted about two hours, all disappearing spontaneously. She had never sought medical assistance, for her mother had had similar experiences in her youth, and informed the patient that they were normal accompaniments of menstruation.

At the time of observation the heart rate was about 200 per minute, regular and easily controlled by ocular pressure. It recurred in a few minutes and was finally stopped by sitting on the patient's abdomen for twenty minutes. The urine was found to be slightly acid immediately after the symptoms disappeared, 5.3 c.c. N/10 sodium hydroxide neutralizing 100 c.c. of urine. The following morning 16 c.c. N/10 hydrochloric acid were necessary to neutralize 100 c.c. of the alkaline urine. Daily urine specimens for the next twenty-three days were slightly acid or alkaline, the amount of N/10 hydrochloric acid or sodium hydroxide ranging from 6.1 to 4.6 c.c. per 100 c.c. urine, respectively. On the 24th day urine was again alkaline, requiring 21 c.c. N/10 hydrochloric acid per 100 c.c., and on the morning of the 25th day requiring 25.6 c.c. N/10 hydrochloric acid. That afternoon the patient developed a paroxysmal tachycardia, which was controlled by a dram of dilute hydrochloric acid and ocular pressure.

The patient was put on a high-fat diet and given 10 drops of dilute hydrochloric acid before meals three times a day. Weekly urine specimens during the next month revealed an acidity corresponding to 24 c.c. N/10 sodium hydroxide per 100 c.c. No attacks were experienced during the next three catamenia, at which time the patient left the city and nothing has been heard from her since.

CASE No. 3.

A. L., a young single man aged 25, was seen in December, 1930. He felt perfectly well at the time of consultation but complained of infrequent attacks of palpitation, breathlessness and weakness which came on suddenly and disappeared after a few minutes. For five years he had worked as a clerk in an office and the attacks had caused him no little embarrassment in his place of business. The past his-

tory was negative except for the exanthemata of childhood.

Physical examination revealed nothing abnormal except dental caries, flabby muscles, and poor posture. The urine was found to be only slightly acid, 100 c.c. requiring 6.15 c.c. N/10 sodium hydroxide for neutralization. X-rays of the teeth showed no apical abscesses and a 7-foot plate of the heart revealed no enlargement.

Exercises were prescribed and the patient advised to stop using tobacco, his average indulgence being about 6 cigarettes a day. Volumetric determination of 24-hour urine specimens collected at weekly intervals the next 3 weeks showed a slight alkalinity. Following a brief attack a few days later alkalinity had increased to a point requiring 13.8 c.c. N/10 hydrochloric acid to neutralize.

A high-fat diet was then prescribed and acetylsalicylic acid, grains 5, three times a day, administered.

In the past four months the patient has been free of symptoms, changed his occupation and regained his self-confidence. Urine tests have shown a consistent acidity of normal grade.

SUMMARY AND CONCLUSIONS

A brief review of the literature on the treatment of paroxysmal tachycardia and three cases are presented.

Urine studies reveal a tendency of these individuals to excrete an alkaline urine at the time of attack.

High-fat diet, supplemented with acids and in one case whiskey, maintained an acid urine and lessened the incidence of attacks of paroxysmal tachycardia.

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DENTISTS NOW TO ADVERTISE ETHICALLY

Group Publicity to Feature Dental Education

The American Dental Association in its National Convention, held at Memphis, Tenn., October 19 to 24, has decided to advertise. This breaks the years' old convention and the official ethics against publicity. However, the advertising will not be in any sense commercial or individualized. Instead, it will be publicity on the highest plane possible, and will be devoted exclusively to dental education.

In a letter to the American Dental Association, sent to its new President, Dr. Martin Dewey, of New York City, President Hoover voiced his sentiments as approving such an advance and modern step and favoring educational publicity, particularly as it will especially benefit the children.

This publicity will be handled by the American Dental Association through a new bureau that has been organized and which will keep the public informed on the care of their teeth, mouth hygiene, proper diet and the prevention of dental troubles. The theme of the publicity will be along the lines of prevention, and, if heeded by the public, will save the people of America millions of dollars in dental bills. Newspapers, magazines, radio and other forms of advertising media will be used in this publicity program, but in no sense will any individual dentists' names be mentioned nor fees quoted.

Such group publicity in the form of dental education was started in Little Rock, Arkansas, in August, 1930, and brought such favorable response and comments from leading dentists, educators and the press that it was decided after such a test to allow the dentists of the country to resort to similar publicity in a likewise ethical manner.

The dentists of the country have taken a forward step, the returns of which publicity will be incalculable, and thus will live up to their ethics of doing everything they can to aid humanity along the lines of dentistry.

"Prevention" as a theme is an unselfish one, and the various messages that will be presented will make the public more dental-minded and cause them to stop neglecting their teeth, and, consequently, their health. —*News story released by the American Dental Association.*

THE COMMON COLD

The United States Public Health Service has published a short treatise on The Common Cold which doctors should have to give to patients. Although there is nothing new from a scientific standpoint the rules for personal behavior are so clearly put that this publication should either be distributed to the laity or doctors should use it as a sermon to be preached to their clientele. The simple technic of personal care should be emphasized by doctors because the statement that colds are contagious is not enough and that is the sum and substance of most of the teaching respecting the health problem of colds.

ACCIDENTS AMONG YOUNG PEOPLE

According to a statement by the United States Public Health Service, accidents rank as the leading causes of death in the age group between 5 and 19 years. Of these the automobile is responsible for about one-third. In this group statistics show that the rate is 15 per cent. less for girls than for boys.

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL
ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 17471

THREE WEEKS' DYSPNEA

MEDICAL DEPARTMENT

A Finnish editor fifty-one years old entered through the Emergency Ward on July 11 complaining of shortness of breath of three weeks' duration.

He was very well until three years before admission, when he began to have dyspnea on climbing hills and an occasional feeling of fullness in the epigastrium, with a substernal choking sensation usually following exertion and associated with dyspnea, very occasionally following meals, usually relieved by rest and rarely partially relieved by milk or soda. These symptoms gradually increased for two years. Eleven months before admission while walking uphill he had sudden griping pain starting in his abdomen, spreading up over his chest and stopping beneath his clavicles, giving a sense of great oppression over the chest, then spreading over his shoulders. When he lay down the pain immediately stopped, lasting about one minute. He got up and walked on, feeling rather weak. After this he had attacks of varying degrees of severity, several times a week only about one a week forcing him to stop and rest. After two weeks he went to a dispensary and later to a doctor, who gave him digitalis for three months. He was able to work with care until April, four months before admission, when three hours after shoveling snow he had dyspnea and an attack of the usual pain which lasted all night. While coughing he raised about 10 cubic centimeters of blood. He stayed in a hospital three weeks with pain and dyspnea all the time, but steadily decreasing. He then rested in bed for a month with no symptoms but weakness. He then stayed in the country, keeping very quiet, and remained symptom-free until three weeks before admission. Then twenty-four hours after a Finnish steam bath, in which there is no exercise or rubbing, while lying on his back on the beach he became orthopneic. He was in the habit of taking such baths every two or three months and had one two months earlier without bad effects. The orthopnea continued all night. The next day he felt well. He kept very quiet until ten days before admission, when he had a similar attack while walking, lasting half the night. A physician gave him digitalis, which he had taken up to admission. He continued to be orthopneic almost every night and sometimes dyspneic during the day without pain and with very slight palpitation. He coughed only slightly. He had lost 20 pounds in the past year. He thought worry over his very responsible work had been one cause of his symptoms.

The family history is not significant so far as known.

He had measles at seven years, typhoid at ten, malaria (?) at sixteen. For ten years he had had night sweats about once a week except for the past year. Beginning four years before admission he had frontal and occipital headache for two years, stopping when he ate less meat. Three years before admission he felt palpitation continuously in his right ear for two months. He urinated three times at night. He had a nervous temperament.

Clinical examination showed a man with evidence of loss of much weight, lying on one pillow, with slightly labored breathing and an apprehensive expression. The skin was pale and sallow. The tonsils showed chronic inflammation. The heart was greatly enlarged to the left. The apex impulse was seen and felt in the fifth space 9.5 centimeters to the left of midsternum. The left border of dullness was 11 centimeters, the midclavicular line 9 centimeters. The supracardiac dullness was 8.5 centimeters. The right border is not recorded. The action was rapid, the rhythm regular. There was a prolonged blowing soft systolic murmur at the apex. The first sound at the apex was obliterated by the murmur. The aortic second sound was thumping and accentuated. The pulses were normal. The radials were compressible, the brachials pulsating but not tortuous. There was pulsation in the vessels of the neck. The blood pressure was 150/100 to 235/162. An electrocardiogram showed normal rhythm, rate 90, inverted T_3. The lungs were clear in front. At the left base there was dullness with scattered moist râles, at the right base more extensive dullness with râles, diminished breath sounds and absent tactile fremitus. The liver edge was felt one to two centimeters below the costal margin. The extremities, pupils and reflexes were normal. Examination of the fundi showed fullness of the vessels, the arteries small and straight, scattered lineal hemorrhages.

The urine was normal in amount. The specific gravity was 1.018 to 1.020. There was a large trace to the slightest possible trace of albumin at six of seven examinations. The sediment showed rare to occasional white cells at six examinations, rare red cells at the last, occasional granular or hyalin casts at four. The renal function was 20 per cent. Blood examination showed 7,500 white cells, 61 per cent polynuclears, hemoglobin 70 per cent, reds 4,600,000, smear normal. A Hinton test was negative. The non-protein nitrogen was 35 to 41 milligrams.

The temperature was 97.5° to 99.5° until August 2. Then there was a steady rise to 108°. The pulse was 75 to 118, with a terminal rise to 160. The respirations were 20 to 49.

The patient responded very well to rest in bed, theocin and digitalis. Twice during the first twelve days he was quite dyspneic. As he became discouraged he was allowed to be up for a short time twice a day beginning July 27. He went rapidly downhill during the next few days and was irrational much of the time. On August 3 he became very irrational and excited, then suddenly fell into a mild semidelirious coma with Cheyne-Stokes respiration and rising temperature. By noon of the following day he appeared moribund, though during a few periods of semiconsciousness he seemed to try to rouse himself and speak. As there was some question of settlement of his will and the prognosis seemed to be a matter of minutes to a few hours, venesection was done, 500 cubic centimeters of blood withdrawn and a grain and a half of caffein given subcutaneously. He returned to consciousness and recognized his family but was unable to talk. One cubic centimeter of coramin was given subcutaneously. With this he improved still more, but still could not articulate and soon relapsed into semicoma. On August 5 he died.

Clinical Discussion

BY RICHARD C. CABOT, M.D.

NOTES ON THE HISTORY

That pain he had eleven months before admission is cardiac pain in all probability, of the type somewhat worse than an ordinary anginal attack but not quite so long as we get with most cases of blocked coronary.

The attack of pain in April lasted all night, therefore it is not to be called angina. It is much more like coronary disease with infarction.

I cannot make anything of the night sweats.

I suppose "palpitation in his ear" means he felt a beating or a rhythmic sensation of some kind, just what I do not know.

NOTES ON THE PHYSICAL EXAMINATION

Irrationality is quite common in cardiac cases.

Venesection is perfectly proper treatment when one is trying to enable a man to get along long enough to make a will.

DIFFERENTIAL DIAGNOSIS

We are dealing with a cardiac case. He has had cardiac symptoms throughout, nothing else. There is the systolic murmur at the apex which we can get with any poor working heart. It does not necessarily mean any valve lesion. I should doubt if there was any. The story is one of coronary disease. I think we should say that he had a hypertrophied and dilated heart of the hypertensive type with coronary disease showing obliteration of one or more arteries and probably infarcts old or new.

The main discussible question is about the kidney. We can say that he probably had the kidney of hypertension. That is a safe way out of the difficulty. We do not have to say anything about nephritis. Anybody with hypertension for any length of time seems to have a definite histological lesion associated with that. But it seems to me he had a very decent kidney function. We have no reason to suppose he would have died of his kidney trouble. It is arbitrary whether we say "nephritis" or not.

The rest of the lesions I suppose were due to passive congestion.

A STUDENT: What was the immediate cause of death?

DR. CABOT: It may have been a fresh infarction; it may have been a pulmonary embolism, but we have no evidence of it here. I think he died a cardiac death.

A STUDENT: How about the temperature?

DR. CABOT: That is a good point. The commonest cause for such fever when there are no particular signs is bronchopneumonia. I say that on general principles. He might have pericarditis in connection with cardiac infarction, if he had infarction.

A STUDENT: Would he go into coma with bronchopneumonia?

DR. CABOT: Yes.

A STUDENT: When the heart fails like that could the kidney fail simultaneously, giving uremia and temperature?

DR. CABOT: Yes, heart failure can precipitate uremia, but there is no rule for temperature in uremia. It can be high or low or normal. The steady rise you see does not sound like merely a terminal or uremic event. He may as you suggest have had uremia partly due to heart failure, but I do not think we have good evidence of it. His symptoms are not uremic symptoms.

A STUDENT: If the coma were due to bronchopneumonia would he have Cheyne-Stokes breathing?

DR. CABOT: Yes, perfectly well, from the kidney, from the heart, or both. We get Cheyne-Stokes breathing from any type of failing heart.

CLINICAL DIAGNOSES (FROM HOSPITAL RECORD)

Essential hypertension.
Hypertensive heart disease.
Congestive failure.
Bronchopneumonia.

DR. RICHARD C. CABOT'S DIAGNOSES

Hypertensive heart disease.
Hypertrophy and dilatation of the heart.
Coronary occlusion.

Probably infarcts of the heart.
The kidneys of hypertension.
Passive congestion, general.
Bronchopneumonia.

ANATOMIC DIAGNOSES

1. *Primary disease.*

(Hypertensive heart disease.)
Cardiac infarction.
Coronary occlusion.

2. *Secondary or terminal lesions.*

Hypertrophy and dilatation of the heart.
Acute bacterial endocarditis of the mitral valve.
Infarcts of the lungs.
Bronchopneumonia.

3. *Historical landmarks.*

Arteriolar sclerosis of the kidneys.

PATHOLOGIC DISCUSSION

DR. TRACY B. MALLORY: This man had coronary occlusion and cardiac infarction; in fact we found two points of occlusion, one in the posterior branch of the right coronary and one in the circumflex branch of the left, and two areas of infarction, an old one and a fresh one side by side. The heart was considerably hypertrophied, weighing 750 grams. The one surprising feature of the case was an acute bacterial endocarditis of the mitral valve which probably had a relation to the murmur and the temperature.

In the lungs were a number of small infarcts and also patches of bronchopneumonia.

The other organs showed passive congestion, and the kidneys showed a slight degree of arteriolar sclerosis.

CASE 17472

GENERAL PERITONITIS, CAUSE UNKNOWN

SURGICAL DEPARTMENT

PRESENTATION OF CASE

DR. HENRY H. FAXON: We got a very uncertain history from this man, who was a retired American sailor, seventy-five years old, and a widower. He entered through the Emergency Ward complaining of intermittent abdominal pain of six weeks' duration. He was practically comatose at the time, so that we could not elicit much history. We did find out that he had been jaundiced off and on for fifteen days before that. The jaundice had become considerably less marked at the time when he came into the Emergency Ward, although it was still present. Cholecystectomy and appendectomy had been done in this hospital five years previously. An interesting thing is that at the time of this operation the symptoms were apparently characteristic of gall bladder disease, but at the time of operation Dr. Richardson had some hesitancy about taking the gall bladder out because apparently grossly it was not especially abnormal. He took out the appendix and the gall bladder. The gall bladder was found to contain a few small stones. Reading between the lines of the history, I think they expected to find more striking gross pathology than was found in the exploratory operation.

The patient had come to the Outpatient Department about once a year for follow-up notes ever since that cholecystectomy and appendectomy five years before. At each one of those visits there had been a note made that he was still having vague upper abdominal distress, the first time four years ago, apparently not marked. However, three years ago the person who saw him noted the previous observation and thought that a gastro-intestinal series was indicated. The record is vague as to whether it was done. I could not find any report of it.

The immediate cause of his coming into the hospital the second time was apparently severe abdominal pain which had been present twenty-four or thirty-six hours. He had vomited more or less, but again our story is rather scanty and it is hard to tell whether he vomited much.

When he came into the Emergency Ward he did not vomit and his stomach was not distended, but he was very much dehydrated and acidotic and the abdomen was practically board-like. There was slightly more tenderness on the left side than on the right, and more in the upper abdomen than in the lower. His temperature was slightly over 100° and his white blood cell count was 14,000. Those were the main facts we had to go on.

We had an x-ray examination made with the idea of seeing if it could show any free gas in the peritoneal cavity. The examination was rather inconclusive. There was no obliteration of the liver dullness that I could make out.

It finally came down to the fact that he obviously had an acute abdomen. All I could say before operation was that he had general peritonitis, cause unknown. It seemed to me that it fitted the picture of a perforated viscus as much as anything else. We considered pancreatitis; we considered perforated gastric ulcer. We went into the history of luetic background and lead. We explored with a preoperative diagnosis of general peritonitis.

DR. CHESTER M. JONES: Was he jaundiced at the time of operation?

DR. FAXON: Slightly. Have you any suggestions as to what we might have done before we operated?

DR. JONES: I should think pancreatitis would be a perfectly good working diagnosis.

DR. FAXON: The history seemed to me too long for acute pancreatitis.

DR. JONES: I meant the subacute type.

DR. FAXON: He seemed almost too sick for the subacute type. Two of the men present however put that as the probable diagnosis.

DR. JONES: He may have had a stone in the common duct in the first place to contribute to the picture.

DR. FAXON: That is very true.

We made a left upper abdominal incision. The abdomen was full of material that looked to me as much like gastric contents as anything else. It was not bile tinged. There was no foul odor. We could not demonstrate any free gas when we opened the peritoneum. The site of his previous operation showed marked adhesions up into the bed of the gall bladder and extending down under his scar. There were a good many adhesions, so that it was impossible to expose the duodenum with safety. We explored the stomach, which we found to be negative, and we explored the abdomen as thoroughly as we could to find a perforation in the stomach or the duodenum or some other cause for his general peritonitis. He was in poor condition, and after we had done what we felt was all he could stand we put a drain into the pelvis and the left flank and closed the wound with the cause of the peritonitis still unknown. I felt that the adhesions around his duodenum probably hid the site of perforation, but I felt that I should do more harm than good in carrying out any extensive lysis of these.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

General peritonitis, cause undetermined.

ANATOMIC DIAGNOSES

Portal thrombosis.
Pylephlebitis.
Subdiaphragmatic abscess.
General peritonitis.
Stones in the common bile duct.
Chronic pancreatitis, slight.
Arteriosclerosis.
Thrombi in the aorta.
Infarct of the heart.
Thrombosis in the vena cava.
Pulmonary embolus.
Bronchopneumonia.
Pulmonary edema.
Atelectasis.
Pericarditis.
Vascular nephritis.
Polyps of the sigmoid.
Diverticula of the ileum.

PATHOLOGIC DISCUSSION

DR. TRACY B. MALLORY: Autopsy showed an unusually complicated pathologic picture. Like so many other men of seventy-five, he had a dozen different ailments, though there is no question but that peritonitis was the actual precipitating factor in his death. That turned out to be secondary to something that had not been considered at all in the preoperative diagnosis, I take it. We happen to have had two other cases during the past year that were almost identical with this, both following chronic sepsis in the biliary tract over a period of years. He had developed a portal thrombosis with pylephlebitis and a small subdiaphragmatic abscess between the left and right lobes, a little more over to the left than the right. At autopsy this abscess seemed to be pretty well walled off, but I think there is little doubt that the peritonitis had developed as an extension from it. We found two stones still in the bile duct. There was a slight degree of chronic pancreatitis, though I think that in all probability it did not play any rôle in the symptomatology.

He had an extreme degree of arteriosclerosis, with numerous thrombi of pretty good size in the aorta. He had an infarct of the heart. He had thromboses partially occluding the vena cava as well as the portal vein, and a pulmonary embolus and bronchopneumonia and pericarditis and vascular nephritis and polyps in his sigmoid and diverticula in his ileum, and the list goes on.

The case is very interesting to me because it makes the third case we have had within a relatively short period of definite pylephlebitis associated, so far as we can make out, with sepsis in the biliary tract rather than sepsis elsewhere in the abdomen, as in the appendix, which is considered a much more classical source of pylephlebitis.

DR. JONES: It is something like Case 17411.* Do you think it was due to the fact that he had pus in the abdominal cavity?

DR. MALLORY: Yes. We have several possibilities there. One of the house officers was afterwards able to get a somewhat better history than they could get in the Emergency Ward before the patient was operated on. As that history was given to me it sounded like recurrent gall stone attacks over a period of five years. He had one of these attacks every few months, each associated with marked pain, vomiting and nausea, with jaundice lasting for a week after each attack and then clearing up. It was only after the family had been very carefully questioned later on that most of that was elicited.

DR. GEORGE W. HOLMES: In view of what we know now it is interesting to look at the abdominal films again. The failure of the films to show free air in the abdominal cavity is of course due to the fact that he did not have a perforated viscus. I wonder if we might have given that more weight at the time.

DR. MALLORY: Another point that is of interest is the fact that this man had a pretty typical history of gall bladder disease five years ago. He was operated on by an excellent surgeon and

*Published October 8, 1931.

the gall bladder was found not to be grossly abnormal, though after it was removed they found a few stones in it. The common duct was not explored. There is a recent article* by Dr. Clute of the Lahey Clinic regarding the frequency with which they have found common duct stones since they began to explore the duct more often. The number of common duct stones has doubled, just in proportion to the increased number of explorations done. It is possible that that point ought to be given more consideration. Where you have a typical history perhaps you ought not to stop until you have explored the common duct.

DR. MAURICE FREMONT-SMITH: How much risk is there in exploring the common duct? Is it not true that the operative mortality is considerably increased when the common duct is opened?

DR. EDWARD D. CHURCHILL: I cannot say on the basis of available statistics. The patients in whom the common duct is explored now are usually the more seriously sick ones, so that mortality figures may well give a distorted view.

*Howard M. Clute, Common Duct Stones. J. A. M. A. Nov. 22, 1930, 93:1568.

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THE PROBLEM OF TRANSPERITONEAL IMPLANTATION OF MALIGNANT OVARIAN TUMORS

OFTEN the apparently obvious has been casually and tacitly accepted even while the exact mechanics of a given process remains highly uncertain. In as long known and well studied a phenomenon as the studding of the peritoneum with masses of tumor tissue, secondary to primary ovarian tumors, the exact course of events by which the metastases become established has been more or less ignored. While metastasis has been generally recognized as by direct implantation it has been considered by some as distribution by subperitoneal lymphatic channels—this in spite of almost invariable localization of the tumor implants on the peritoneal surfaces; often the peritoneal surface of the diaphragm while the pleural and pericardial surfaces are free from involvement.

A valuable contribution to our knowledge of the process is a logical sequence to the studies of Sampson[1] on endometrial implantation. He has taken this problem of metastasis and has established the exact mechanism whereby the fragments of tumor cells succeed in establishing themselves on the peritoneum[2]. The initial process is the irritation of the peritoneum by the presence of free nodules of tumor cells, which act more or less as inert foreign bodies. The immediate response is an exudation of fibrin which serves to fix the tumor cells at the site of irritation. Next, organization of the fibrin takes place, which brings fibroblasts and capillaries into intimate contact with the tumor cells which continue to grow in the favorable environment created by the natural conditions of the peritoneum. With steady growth of the tumor cells the fibroblasts and capillaries are appropriated by the tumor as stroma and thorough establishment of the tumor nodule in the site of localization is assured. Sampson has succeeded in following every step of this process from the detection of free floating masses of tumor cells in the peritoneal cavity to the development of the metastatic nodule. This demonstration very definitely establishes direct transperitoneal implantation as the common mechanism of metastasis in malignant ovarian tumors.

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2 Sampson, John A.: Am. J. Path., 7:423, 1931.

CONSTANTINE VON ECONOMO

THE passing of Professor von Economo, the distinguished Vienna neurologist, October 21, 1931, at the early age of fifty-five, is a very distinct loss to medicine. He is best remembered for his early and accurate description of a disease of the nervous system which he called "encephalitis lethargica". His patients were observed toward the end of 1916 and Economo's first paper was given before the Vienna Society of Psychiatry and Neurology on April 17, 1917. In his report he pointed out both the clinical and the pathological aspects of the disease and demonstrated conclusively that the medical world was faced with a new, heretofore unknown, epidemic infectious disease of the nervous system. Since that time extensive research on the subject has been undertaken, for the disease soon appeared throughout the Continent of Europe, then in the British Isles, in America, and thus around the entire globe. Although the early cases as described by Economo were largely of the lethargic type, subsequent observations have broadened the original description into a disease of protein manifestation. Economo's final summary of epidemic encephalitis has recently been published in English by the Oxford University Press (1931).

Besides his description of encephalitis, Economo also did excellent work, of lasting value, on the finer architecture of the cerebral cortex. In addition to his medical interests he was known as one of the pioneer aviators of

Austria and at the time of his death was honorary president of the Austrian Aeronautical Club. In 1929 he visited the United States and is pleasantly remembered by those who heard his excellent lectures in New York and elsewhere. His position in medical history is secure for he saw the importance of a new disease and pointed out to the medical world its main characteristics.

THE ANNUAL CHRISTMAS SEAL SALE

THE several tuberculosis organizations throughout the State are perfecting plans for the distribution of the seals which will be sold to all persons who are disposed to help in this method of raising funds for the prosecution of antituberculosis work.

The more common method in vogue at the present time is to distribute seals through the mail to those who are supposed to have an interest in the activities of the National and State organizations.

This annual effort has become an established procedure and has been very generally endorsed. "The Great White Plague" has lost its position at the head of the mortality rates and is gradually becoming less and less a menace to humanity, but although not so commanding as formerly it destroys thousands every year in the most productive period of human life.

The continuous warfare waged against this disease has enabled statisticians to predict a definite decline in each ten-year period. This prognosis has been verified.

The method employed in antituberculosis work has been intelligently and continuously employed for more than forty years and stands as a demonstration of effective preventive health measures.

The success of antituberculosis effort should encourage those interested in other fields.

The medical profession can add to the success of the seal sale by a suggestion here and there to their patients to buy as many seals as circumstances will warrant.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

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HICKS, JAMES B. B.S., M.D. Johns Hopkins University School of Medicine 1924. Urologist for the Lahey Clinic. His subject is "Cystinuria Complicated by Cystine Calculi with a Case Report." Page 1001. Address: 605 Commonwealth Avenue, Boston.

STUART, HAROLD C. Litt.B., M.D. Columbia University College of Physicians and Surgeons, 1918. Assistant Professor of Pediatrics and Child Hygiene, Harvard University Assistant Physician at Children's Hospital, Boston. Visiting Physician at Infants' Hospital, Boston. Vice-Chairman of the Committee on Growth and Development of the White House Conference on Child Health and Protection. His subject is "The Findings of the Committee on Growth and Development of the White House Conference on Child Health and Protection." Page 1004. Address: 55 Van Dyke Street, Boston.

HEALY, JAMES C. Ph.G., M.D. Tufts College Medical School 1927. Junior Visiting Physician, Boston City Hospital. Assistant Visiting Physician, Long Island Hospital. Visiting Physician to Out-Patients, Carney Hospital. Instructor in Medicine and Pharmacology, Tufts College Medical School. His subject is "Treatment of Paroxysmal Tachycardia." Page 1010. Address: 416 Huntington Avenue, Boston.

MISCELLANY

TO FELLOWS OF THE MASSACHUSETTS MEDICAL SOCIETY

In the *Journal* of October 29, p. 883, the Treasurer of the Massachusetts Medical Society made an especially attractive offer of the remaining copies of Dr. Viets' *Brief History of Medicine in Massachusetts*. As that notice may have been overlooked by some of the Fellows of the Society, the Treasurer wishes again to call attention to it; and to offer to Fellows, at the greatly reduced price of $1.25, this interesting volume. The Treasurer hopes that many more of our Fellows will avail themselves of this opportunity. Orders will be received by the Treasurer, 8 The Fenway, Boston; and copies will be sent, postpaid, on receipt of checks to cover.

CHARLES S. BUTLER, *Treasurer*.

8 The Fenway, Boston.

THE FAULKNER HOSPITAL CAMPAIGN FOR FIVE HUNDRED THOUSAND DOLLARS

One hundred and sixty-eight physicians of Metropolitan Boston have joined the Doctors' Committee in the Faulkner Hospital's campaign for $500,000 to remove indebtedness, it has been announced by Dr. Edward L. Young, Jr., chairman of this committee and surgeon on the Hospital's staff. Approximately half of this group attended the committee's first meeting, a luncheon held at the University Club of Boston on October 29.

At this meeting, presided over by Dr. Young, honor was paid to the five remaining members of the Faulkner's original staff by the Hon. Andrew J. Peters, president of the Hospital. When the Faulkner Hospital was opened in 1903, ten members constituted its staff. Four of this group have died and only one has resigned. The five who have served continuously on the Hospital's staff are Dr. Franklin G. Balch, consulting surgeon; Dr. Arthur N. Broughton, assistant surgeon; Dr. William W. Howell, pediatrician; Dr. Henry Jackson, consulting physician, and Dr. John S. H. Leard, medical staff. All of these men are graduates of Harvard College and of the Harvard Medical School except Dr. Leard, who was graduated from the University of Pennsylvania. Their combined service to Boston aggregates 188 years.

The Endorsing Committee for the Faulkner Hospital's campaign consists of 60 leaders in Boston's civic and philanthropic life. Its chairman is Mr. George W. Mitton, head of the Jordan Marsh Company. A total of nearly 800 people have become concerned in the effort of the Faulkner Hospital to clear itself of indebtedness. The campaign entered its intensive period with the dinner at the Hotel Somerset, Boston, on Tuesday, November 17.

The necessity for the Faulkner Hospital's campaign is said by its sponsors to be due to three factors: (1) the Hospital has had to grow considerably to keep abreast of the hospital needs of one of Boston's fastest-growing districts; (2) the Faulkner's growth has been accompanied by almost no growth in the number of its most generous friends, and (3) the indebtedness resulting from these two factors has now reached the acute stage.

Stated very briefly, the City and the Metropolitan District of Boston in only 29 years have surrounded the Faulkner Hospital, which was originally at the city limits. Boston's growth has made the Faulkner Hospital the health center of a rapidly-growing urban district.

While the City of Boston has grown 4.6 per cent. in population since 1915, the southwest part of the city, which is mainly served by the Faulkner Hospital, has grown nearly 70 per cent. The entire southwest portion of Boston now contains approximately 80,000 residents. From this area sixty per cent. of the Hospital's patients come. The remaining forty per cent. come from all over Metropolitan Boston.

On account of the increasing growth of the Hospital, its trustees in 1924 invited a thorough survey by Dr. Frederic A. Washburn, Director of the Massachusetts General Hospital. He advised the Faulkner to "keep ahead of the needs of the community —that, otherwise, small hospitals would be started and the resources of the community would be wasted because of the division of effort and money".

With its direction thus clearly pointed, the Faulkner Hospital at once purchased eight additional acres of land adjoining its Centre Street front, to guarantee room for future growth. On part of this land was erected, in 1926, the new Nurses' Home. Since in the three years 1927-29 the Faulkner's 75 beds were used to 96 per cent. of capacity (80 per cent. is normal in hospital practice), the Hospital in 1929 began construction of the new Surgical Wing, using all unrestricted funds on hand and borrowing $470,000 in order thus to serve the public.

This unit, connected with the older buildings, doubled the Hospital's bed capacity. It also made available to the community the most modern equipment for surgery, preoperative diagnosis, and laboratory analysis. The new building contains three general operating rooms, three nose and throat operating rooms, and one plaster room. It contains also chemical, pathological, and bacteriological laboratories. In addition, there is an enlarged x-ray and fluoroscopy department, an electrocardiogram room, a cystoscopy department, and an enlarged metabolism service.

After the new Surgical Wing was opened in April, 1930, the Hospital completely modernized its older buildings. Meanwhile it had also added a service building and a new, economical heating plant.

In brief, the Hospital today has a plant consisting of eight buildings and fifteen acres of land. The total value of its property has increased from $668,156 to $1,409,540 since 1924. Its regular staff includes 31 physicians, and 79 others constitute the Courtesy Staff. Its chief staff officers include: Consulting Surgeon, Dr. Franklin G. Balch; Consulting Physician, Dr. Henry Jackson; Surgeon, Dr. Edward L. Young, Jr. The Superintendent is Miss Frances C. Ladd. To bulwark the work of the staff, the Hospital maintains its own Nurses Training School, from which 237 young women have been graduated.

To sum up, the Faulkner Hospital has grown as Boston has grown. A glance at a map will show that the City must probably continue to expand toward the southwest. Located precisely in the path of this growth, the Faulkner Hospital has prepared itself to give the increasing service that this growth will necessitate; but the Hospital has building indebtedness of $470,000 and accumulated deficits from 1929 and 1930 amounting to $23,564. These are due to necessary interest payments of nearly $23,000 a year on the Hospital's obligations. It is to clear itself of this $500,000 indebtedness that the Faulkner Hospital is making its first public appeal to the citizens of Boston.

A COMPLIMENTARY DINNER TO DR. FRANKLIN S. NEWELL

On Saturday evening, November the seventh, a dinner was given at the Country Club, Brookline, in honor of Dr. Franklin S. Newell, whose retirement as Professor of Clinical Obstetrics at the Harvard Medical School, has become effective with the current academic year.

Many of the seventy guests present were graduates of the Boston Lying-In Hospital, others were contemporary colleagues and personal friends of Dr. Newell; and all came with the thought of honoring a great clinical teacher. This thought was happily phrased by Dr. Harvey Cushing, who, as toastmaster, with characteristic grace and eloquence, compared the Boston Lying-In Hospital with the Dublin Rotunda as one of the cornerstones of obstetric thought and teaching throughout the world, and paid tribute to the distinguished work of the guest of honor in his long career as its head.

The span of professional life is short, at best, but among its greatest rewards is the privilege of contact and association with those who by their teaching and example set a standard for their followers to emulate. That this feeling is present among those who owe their training to Dr. Newell was made evident by the sincerely affectionate testimonial of November the seventh.

A RADIO MESSAGE PREPARED AND SPONSORED BY THE COMMITTEE ON PUBLIC EDUCATION OF THE MASSACHUSETTS MEDICAL SOCIETY FOR THE DEPARTMENT OF PUBLIC HEALTH

High Blood Pressure*

A person cannot go very far these days without encountering a discussion of blood pressure—in the newspaper, magazine, club, or over the radio, and "How is your blood pressure?" is a frequent question at the bridge table. Together with its popularity, however, there has developed a fear of the disease, a fear that is born of that "little knowledge that is dangerous". Very few laymen know what blood pressure is, what produces it, how long high blood pressure lasts, and many other simple facts the understanding of which should remove a great deal of the fear associated with the words "high blood pressure". I hope in the next fifteen minutes to present some of these simple facts to you.

WHAT BLOOD PRESSURE IS

"Have I any blood pressure today?" is a frequent question that patients ask. The answer is that everybody has a blood pressure so long as he is alive. Without this pressure the blood would not circulate through the body. Blood pressure is the force of the blood, which is seen, for example, when you cut your finger and the blood spurts out. Blood pressure is like the force of the water coming out of a fireman's hose. This force or pressure of the blood is caused by the pumping of the heart which forces the blood through the arteries all over the body. It is this force of the blood which varies in people with normal, high or low blood pressure.

NORMAL BLOOD PRESSURE

The normal pressure of the blood does not change very much from youth to old age. The old rule about "your age plus one hundred" is no longer used. It is unreasonable to expect your physician to say that such and such a figure should be your blood pressure, because the normal blood pressure is a very variable force at all times of the day and under different conditions from moment to moment. When you go to sleep your blood pressure becomes very much lower. If you exercise it may temporarily become higher. On a very warm day or after a warm bath it may become lower. If you flare up in anger it may temporarily rise. It is lower in the morning when you first get around than it is after a day full of activity. These are but a few of the numberless things that make your normal blood pressure change during the course of the twenty-four hours to such an extent that at one time of the day you may have a low and at some other time a high blood pressure. It is for this reason that you cannot expect your physician to say exactly what your blood pressure should be.

HIGH BLOOD PRESSURE

It is only when your blood pressure as found on several visits to your physician, tends to remain at a higher or lower level, that a true suspicion develops that we are dealing with an abnormal condition.

To understand how the blood pressure becomes high, remember first how I explained the normal blood pressure. If you will again picture water coming out of the nozzle of a hose, you will see that if the nozzle is turned so as to make its opening very small, the water will come out with much greater force or pressure. In people with high blood pressure the small arteries are of smaller than normal calibre, and, therefore, as in the case of the hose and nozzle, the blood has a greater pressure. In other words high blood pressure is due to some unknown condition which makes the small arteries narrower than normal, or as we say makes them contract or constrict. High blood pressure is not due to heart trouble as many people think.

One of the most characteristic things about high blood pressure (and I speak now of the most common kind called by physicians essential hypertension) is the variability of the blood pressure under different conditions. During relaxation it becomes lower, and during sleep it is often normal. After a hard day or an exciting event it is higher. In other words it is very much like the variations of the normal blood pressure, only in high blood pressure, the pressure tends to remain above the normal.

You will, of course, notice that I have not said

*Broadcast November 5, 1931—Station WBZA—4:50 P. M.

anything about the actual figures of blood pressure, and by now you probably realize how impossible it is for a physician to tell you your exact blood pressure. It might be 200 when you first come into the office, 160 after you rest awhile, 140 when you return to the calmness of your home, and 100 when you are asleep. It should be sufficient, therefore, if your physician simply tells you that you have a normal blood pressure, that you tend to have a high blood pressure, or that in general your blood pressure is better. Do not ask him for the individual readings of your blood pressure which by themselves are necessarily meaningless to you. The statement that patients make—that if they know the height of their blood pressure, they would know how to care for themselves is wrong because, since their blood pressure is always going up and down, they should always take care of themselves to the same degree regardless of what the pressure is at any one moment.

Most uninformed people think that they are going to die very shortly just because their blood pressure is found to be high. This is distinctly an incorrect idea. Except in a very few patients, high blood pressure is a chronic disease which usually lasts for many years. Many patients with undoubted high blood pressure state that their physicians told them they had the disease 15 to 20 years before. In people, therefore, whose arteries are in good condition, the existence of high blood pressure does not prevent a useful life for as much as 20 years or more.

Many patients go through life with a high blood pressure and have no complaints at all. Headaches and dizziness which in the minds of patients are always associated with high blood pressure are very frequently not due to the blood pressure at all but to the unnecessary worry about the blood pressure. These headaches are like those that develop for example after a tremendous financial loss, and the dizziness like that which we experience on looking down from a high place and becoming temporarily alarmed. These headaches and dizzy spells do not by themselves mean "shock" or impending shock as so many patients incorrectly believe. They do indicate, however, that the patient should see his physician and avoid his own incorrect, pessimistic interpretation.

Since the actual cause of the narrowed arteries and increased blood pressure is not known, there is no positive and absolute cure as in diphtheria and some other diseases. There is, however, a great deal that may be done to prolong life for many years. The most important single thing is to lead a more relaxed, nonchalant, and carefree life. Most people with high blood pressure are of a very active nature, either physically or mentally. They are driving their bodies and minds at sixty miles an hour. The thing for them to do is to slow down their pace to twenty miles an hour so that their human machine may last much longer. There are many other personal peculiarities of people with high blood pressure that need to be changed. In general a careful analysis of their entire daily life and activity should be made and a detailed mode of life prescribed. In addition there are various simple, well-known drugs that physicians use to help along at times, and these, of course, should be prescribed only by the physician. All newspaper and magazine advertisements of wonderful remedies and cures for high blood pressure are, of course, not true.

If you wish to be modern and sensible, you ought to have periodic physical examinations no matter how well you feel. Such a preventive measure is especially desirable in families in which a close relative has or had high blood pressure; for, as yet, the only positive fact about the cause of high blood pressure is that it strongly tends to run in families. Since it is also known that high blood pressure frequently commences in early life—not infrequently at twenty years of age—it would seem best that all members of families in which a close relative has or had high blood pressure, should have semi-annual complete examinations. In this way the early discovery of the disease will bring a more adequate treatment and, possibly, a cure.

High blood pressure, therefore, is not a terrible disease. It is a much simpler disease to have than many others; for, by following the advice of your physician, you may lead a long and useful life despite the disease.

THE ADDITION TO THE MATERNITY HOME AND HOSPITAL OF THE FLORENCE CRITTENTON LEAGUE

The new wing of the home and hospital of the Florence Crittenton League of Compassion has been completed, thus making this group of buildings well-fitted for the care of convalescent girls, recent admissions and for girls out of work who need temporary shelter. The cost of these buildings is four hundred thousand dollars, including this last addition, which cost one hundred thousand dollars. The plant is now believed to be the best appointed institution of its kind in the world.

The twentieth annual bazaar in aid of the League is being held this week at the Copley-Plaza. The organization merits the support of the public.

THE CAMBRIDGE TUBERCULOSIS ASSOCIATION HAS CHANGED ITS NAME

At the recent annual meeting of the Cambridge Tuberculosis Association the name of this organization was changed to "Cambridge Tuberculosis and Health Association."

The following named officers were elected: Dr. Hilbert F. Day, president; Mrs. Rufus P. Williams and Dr. Felix McGirr, vice presidents; Miss Louise W. Jackson, secretary, and William D. Swan, Jr., treasurer.

RÉSUMÉ OF COMMUNICABLE DISEASES IN MASSACHUSETTS FOR OCTOBER, 1931

Poliomyelitis has shown a decided decrease from the peak of the outbreak, which occurred in September. The number of cases, however, is still greater than in any other previous year except 1916 and 1927.

Scarlet fever was considerably more prevalent than usual, the number of cases reported being exceeded in only about four years since 1906.

Mumps was likewise quite prevalent, being the highest number of cases reported in any October up to this time.

Diphtheria, on the other hand, has continued its low record established in earlier months of the year, the number of cases reported being the fewest ever reported in October.

Measles is also low, 1926 being the only year since good reporting was begun that there was a fewer number of cases reported in this month.

Lobar pneumonia has likewise established a new low record, with the lowest number of cases since the disease began to be reported.

Typhoid fever continued its low prevalence, 1930 being the only year in which there were fewer cases reported in this month.

Chicken pox has been reported only half as often this month as in any of the last five years.

Pulmonary tuberculosis and other forms of tuberculosis show a decrease over 1930, which was the lowest year up until then.

Whooping cough, German measles, epidemic cerebrospinal meningitis and influenza were reported about as often as was expected.

MONTHLY REPORT OF CERTAIN COMMUNICABLE DISEASES

	Cases in Entire Pop.				Case Rates per 100,000 Pop.		
	Oct. 1931	Oct. 1930	Proso. Index*	Epi. Index†	Oct. 1931	Oct. 1930	Expected Rates‡
All Causes	4470	4279	—	—	104.5	100.6	—
Ant. Polio.	234	170	55	4.3	5.5	4.0	1.3
Diphtheria	196	302	330	.6	4.6	7.1	7.7
Ep. C. S. Men.	8	7	4	2.0	.2	.2	.1
Measles	173	339	677	.3	4.0	8.0	15.8
Pneu. Lobar	189	201	205	.9	4.4	4.7	4.8
Sc. Fever	682	436	598	1.1	15.9	10.2	14.0
Tbc. Pul.	344	351	339	1.0	8.0	8.2	7.9
Ty. Fever	35	34	37	.9	.8	.8	.9
Wh. Cough	323	304	769	.4	7.6	7.1	18.0
Chicken Pox	208	486	—	—	4.9	11.4	—
Ger. Measles	36	41	—	—	.8	1.0	—
Gonorrhea	662	617	—	—	15.5	14.5	—
Influenza	32	11	—	—	.7	.3	—
Mumps	293	99	—	—	6.9	2.3	—
Syphilis	355	371	—	—	8.3	8.7	—
Tbc. Other Forms	34	45	—	—	.8	1.1	—

*This index is an attempt to estimate the number of cases based on the trend during the past years which can be expected to occur, and is for the purpose of comparison with the number of cases which actually did occur.

†This ratio expresses how prevalent the disease is compared with the index mentioned above; 1.0 indicates that the actual number of cases equals the expected number. A larger number means a greater prevalence, and a smaller number a lesser prevalence than expected. Thus, 2.0 would indicate twice the expected number, and .5 half the expected number of cases. The method used to determine the indices is described in the August 18, 1927 issue of the *Boston Medical and Surgical Journal*.

‡Calculated from the Prosodemic Index.

RARE DISEASES

Actinomycosis was reported from Foxboro, 1.

Anterior poliomyelitis was reported from Abington, 1; Agawam, 4; Amherst, 3; Ashburnham, 3; Ashfield, 1; Belchertown, 3; Boston, 17; Braintree, 2; Brockton, 8; Cambridge, 4; Chelsea, 1; Chicopee, 3; Concord, 1; Colrain, 2; Conway, 1; Danvers, 1; Dartmouth, 1; Dighton, 1; Easthampton, 1; Easton, 1; East Brookfield, 1; Erving, 1; Everett, 4; Fall River, 2; Fitchburg, 15; Framingham, 2; Gardner, 3; Gloucester, 2; Grafton, 1; Greenfield, 2; Hadley, 2; Hanover, 3; Haverhill, 3; Holden, 1; Holyoke, 4; Lawrence, 2; Leominster, 9; Lexington, 1; Lowell, 2; Lynnfield, 1; Malden, 2; Marshfield, 1; Medford, 2; Middleboro, 1; Middleton, 1; Millbury, 1; Monson, 6; Natick, 2; Needham, 1; New Bedford, 1; Newburyport, 1; New Marlboro, 2; Newton, 4; Northampton, 10; Orange, 2; Peabody, 1; Petersham, 1; Pittsfield, 2; Provincetown, 3; Quincy, 2; Rehoboth, 1; Russell, 1; Salem, 3; Scituate, 1; Somerville, 2; South Hadley, 1; Springfield, 15; Sunderland, 1; Sutton, 2; Swampscott, 3; Templeton, 1; Waltham, 4; Ware, 4; Warren, 1; Watertown, 3; Wayland, 1; Wenham, 2; Westhampton, 4; Williamsburg, 1; Williamstown, 1; Winthrop, 4; Worcester, 12; total, 234.

Anthrax was reported from Haverhill, 1.

Dysentery was reported from Worcester, 1.

Encephalitis lethargica was reported from Danvers, 1.

Epidemic cerebrospinal meningitis was reported from Boston, 2; Cambridge, 1; Holyoke, 1; Quincy, 1; Salem, 1; Saugus, 1; Somerville, 1; total, 8.

Malaria was reported from Fall River, 1; Worcester, 1; total, 2.

Pellagra was reported from Boston, 1; Northampton, 1; Worcester, 1; total, 3.

Septic sore throat was reported from Beverly, 1; Boston, 6; Fall River, 1; Gardner, 1; Lexington, 1; Lowell, 1; Melrose, 1; New Bedford, 1; Springfield, 1; Templeton, 1; Waltham, 1; Winchester, 1; total, 17.

Tetanus was reported from Boston, 1; Chicopee, 1; East Longmeadow, 1; Groton, 1; Melrose, 1; Natick, 1; Northampton, 1; Saugus, 1; Winthrop, 1; total, 9.

Trachoma was reported from Boston, 2; Springfield, 1; total, 3.

Trichinosis was reported from Framingham, 1.

A REMARKABLE SERIES OF DOCTORS

In recognition of several generations of doctors the town of North Andover recently dedicated a drinking fountain to perpetuate a record of service to humanity by a remarkable line of physicians beginning with John Kittredge in 1630, who was followed by eight generations, all bearing the family name, with an aggregate service covering about three centuries.

The North Andover fountain was given by John Kittredge and his wife, Frances B. Kittredge of Taunton.

SEVENTY YEARS OF SERVICE BY THE NEW ENGLAND HOSPITAL FOR WOMEN AND CHILDREN

An illustrated booklet has been issued by the New England Hospital for Women and Children which gives a brief history of the seventy years of service to the sick by this hospital, which started with ten beds and has this year opened a new unit for children. This brings the group of buildings up to five with accommodations for 270 patients.

This institution was founded by Dr. Marie Zakrzewska, who had a large practice and was generally regarded as a wise physician. The hospital has the honor of having taught Linda Richards, the first trained nurse in America, conferring her diploma in 1873. Another person who added to the reputation of this hospital was Susan Dimock, who became the resident physician in 1866 after obtaining her medical education in Zurich and Vienna. Last year 1267 children were born in the hospital and, since 1862, the total number of births has been nearly 28,000. The hospital depends largely on charitable contributions and is now carrying on a campaign for necessary funds, which should be generously supported.

CORRESPONDENCE

A DISCLAIMER RESPECTING ALLEGED ATTEMPTS TO SECURE LEGISLATION BY THE OSTEOPATHIC SOCIETY

November 9, 1931.

The *New England Journal of Medicine,*
To the Editor:

In the report of the proceedings of the Council of the Massachusetts Medical Society, which you print in your October twenty-seventh, nineteen thirty-one issue, Dr. Thomas J. O'Brien is quoted as saying the following in regard to the plans of the Osteopaths:

"The Osteopaths are going to present a bill requiring the Governor to appoint an Osteopath on the Board of Registration in Medicine. If they do not do that they are going to present a bill to create a board of osteopathy. In nineteen hundred and nine, we turned down the osteopaths, and made them come up to our grade, and now, if you please, they have the legal right to operate, and they have a hospital in Jamaica Plain, and they can do Caesareans, and set fractures and do what they please and our law permits it."

We wish to say that the Massachusetts Osteopathic Society has no knowledge of any such legislative plan. Following Governor Ely's failure to appoint an Osteopath on the Board of Registration in Medicine, it has been suggested that we introduce a clause into the law that will make it clear who may be so appointed, but every effort will be made to avoid so-called class legislation.

We wish also to call to your attention that Osteopaths began to take the examination and became registered physicians in eighteen hundred and ninety-nine, and at the time the Osteopathic Law was passed in nineteen hundred and nine, there were then, about one hundred with the legal rights and privileges of physicians.

It should occasion no particular surprise that after thirty-two years we are able to present a modern hospital dedicated to charitable work for the underprivileged of our Commonwealth, and supplying competent and skillful service in every field which we undertake.

Very respectfully yours,
FRANK M. VAUGHAN,
PERRIN T. WILSON,
Committee of Censors for the Massachusetts Osteopathic Society.

RECENT DEATHS

CARLEY—DR. FREDERIC JAMES CARLEY, for some 25 years a practicing physician in North Attleborough, killed himself in a sensational manner in the garage of his home in that town, November 10, 1931.

Dr. Carley was born in Plymouth in 1878, was graduated from Tufts College Medical School in 1906 and joining the state medical society settled in

New Bedford, moving soon to North Attleborough. He was a past president of the Rotary Club, and of the Y. M. C. A. and an organizer of the Community playground. He was also an Elk, a Mason, a member of the Universalist Church and chairman of the local chapter of the Red Cross. He was a popular club man and had a large practice. He is survived by his widow and by two children, a son and a daughter.

CURRY—Dr. Edmund Farnham Curry died at the Union Hospital, Fall River, of coronary occlusion, October 29, 1931, aged 59.

A native of Winchester he received his M.D. at Harvard Medical School in 1896 and settled in Fall River two years later. He had practiced surgery and was a member of the New England Pediatric Society. Dr. Curry served first as a councilor from the Bristol South District of the Massachusetts Medical Society in 1916-1917. He was a councilor at the time of his death. In 1925 he was president of the Bristol South District Medical Society.

He is survived by his widow and two children.

He was on the Surgical Staff of the Union Hospital and the Fall River General Hospital at the time of his death. He had served as Vice President of the New England Pediatric Society.

He belonged to the Harvard Club of Boston, the Rod and Gun Club and Montauk Gun Club of Fall River.

Early in his practice Dr. Curry did much in securing the production of clean milk for babies. He was admired for his diagnostic keenness, careful and gentle operating and painstaking care.

OBITUARY

LEMUEL FOX WOODWARD, M.D.

Death has taken a Worcester surgeon, long prominent in medical circles in that city, one who was affectionately known as "Dr. Lem". He died at his home, still in active practice, November 12, 1931, of cerebral hemorrhage, at the age of 74. His father was Dr. Rufus Woodward, a practitioner of Worcester who died in 1885 shortly after his son had completed a course of study at the Lawrence Scientific School at Cambridge and at Harvard Medical School. His mother was Jane Fox, also of Worcester. While at college young Woodward rowed a stroke oar in a Harvard crew and going to Worcester he became captain of the Quinsigamond Boat Club, an interest that was near to him throughout life.

Dr. Woodward's grandfather was Dr. Samuel Bayard Woodward (1787-1850), who after making the beginnings of the Hartford Retreat in Hartford, Connecticut, moved to Worcester and established the Worcester Asylum for the Insane, the first hospital in the State for the care of the indigent insane, now known as the Worcester State Hospital.

On receiving his M.D. from Harvard in 1882 and completing terms of service as house officer at the Boston City Hospital and at the Boston Lying-In Hospital "Dr. Lem" opened an office for general practice and soon acquired hospital connections at the Worcester Memorial, Worcester City and St. Vincent hospitals. Later he was visiting surgeon to the first two. When his father died the support of his mother and sister devolved on him and he was busily employed for a series of years; a big body and good physique enabling him to do much hard work day and night. He served his city as chairman of the board of health for ten years, and city physician as well. He was physician to the Worcester jail and to the house of correction, police surgeon and physician to the Firemen's Relief Association. Later in life he devoted more and more attention to life insurance and became chief examiner for the Worcester district of the Metropolitan Life Insurance Company, an examiner for the United Commercial Travelers' Association and for the Travelers Insurance Company. Previous to this he was an active surgeon at his hospitals and was in demand as a consultant and operator far beyond the boundaries of Worcester County.

Dr. Woodward was a Fellow of the Massachusetts Medical Society and served for many years as a councilor from the Worcester District, the society of which elected him president in the year 1909. He was a Fellow of the American College of Surgeons and of the American Medical Association and served as first secretary of the Massachusetts Association of Boards of Health. He was a member of a small dining club composed of some leading practitioners of Worcester that has had a happy existence for a long period of time and he held membership in several of the social clubs of his city.

A friend of the out of doors "Dr. Lem" was always interested in bird life. He maintained feeding trays in winter at the Quinsigamond Boat Club for the benefit of his feathered friends and he knew a majority of the birds of that region both in their summer and winter plumage; he was keen in noting their migrations. He served as president of the Worcester Natural History Society, as had his father before him, and made the society of more use to the community. His garden at the boat club was a constant source of pleasure to him and all of the many aspects of the crops were of constant note and of discussion with his friends.

Dr. Woodward was a well-known figure on the streets of Worcester, where, in the early days of his practice, he drove about in a buggy with a high-spirited horse and in later life in motor cars. He had a commanding presence with his large frame and he had a kindly nod for everyone of a large acquaintance. He was never married and lived with his sister, Miss Jane Woodward, in the brick house on Pearl Street which had been built by their parents. She is his sole immediate survivor.

NOTICES

HARVARD UNIVERSITY MEDICAL SCHOOL

Dr. Henry E. Sigerist, Professor in the History of Medicine at the University of Zurich, will lecture on Civilization and Disease on Monday, November 30, 1931, at 5 P. M., in Amphitheatre C, Harvard Medical School.

This will be the first of a short series of lectures on the History of Medicine.

RADIO HEALTH MESSAGES

Sponsored by the Public Education Committee of the Massachusetts Medical Society and the Massachusetts Department of Public Health.

Courtesy WBZA. Thursdays at 5:17 P. M.

November
26 Fatigue as a Symptom of Disease

December
3 Overweight—Cause and Prevention
10 Can We Prolong Life?
17 Pasteurized or Raw Milk
24 Public Health in the Coming Year
31 Mental Health

STATE HOUSE BROADCAST

Sponsored by the Massachusetts Department of Public Health.

Courtesy WEEI. Fridays at 12:30 P. M.

The various activities of the Department are discussed by the Directors of its nine Divisions.

RADIO HEALTH FORUM

Directed by the Massachusetts Department of Public Health.

Courtesy WEEI. Fridays at 4:50 P. M.

Queries from the public are answered under the sponsorship of the Department.

Questions on Health and Prevention of Disease may be sent to: Radio Health Forum, State Department of Public Health, State House, Boston.

PROGRAM

SPECIAL SERIES OF BROADCASTS

Sponsored by the Massachusetts Department of Public Health. Courtesy WEEI. Mondays at 7:15 P. M.

Health from Zero to Ninety

November
23 Preschool and School Health
Stanley H. Osborn, M.D.
30 Health and the College Student
Mary DeKruif, M.D.

December
7 Health and Safety of the Young Adult
C.-E. A. Winslow, Ph.D.
14 Health At and After the Prime of Life
Herbert L. Lombard, M.D.

WHITE HOUSE CONFERENCE ON CHILD HEALTH AND PROTECTION

Round Tables

December
21 Round Table No. 1—Why Another Conference?
Conducted by George H. Bigelow, M.D.
28 Round Table No. 2—How Much Care Does the Well Child Need?
Conducted by Mr. Frank W. Kiernan

1932
January
4 Round Table No. 3—Health in Education—Education in Health
Conducted by Raymond Patterson, Ph.D.
11 Round Table No. 4—What a Community Owes to Its Children
Conducted by Lila Owen Burbank, M.D.
18 Round Table No. 5—Unto the Least of These
Conducted by Mr. Alfred F. Whitman

REPORTS AND NOTICES OF MEETINGS

THE GEORGE W. GAY LECTURE ON MEDICAL ETHICS

Dr. John M. Birnie of Springfield gave the George W. Gay lecture on medical ethics at the Harvard Medical School on November 4. His subject was "Ethics for the Doctor."

He read and commented upon the code of ethics of the American Medical Association. The code is simply the application of broad ethical principles to the circumstances arising in practice. Dr. Birnie especially warned the students that all other considerations should at all times be subsidiary to the welfare of the patient, that professional secrecy should be guarded at all times, and that any form of the commission plan is entirely unethical.

He concluded his talk with a few suggestions of his own. Above all, the doctor should be a gentleman. This may be defined as "one who has more regard for the rights of others than for his own feelings and for the feelings of others than his own rights".

THE GREATER BOSTON MEDICAL SOCIETY

The Greater Boston Medical Society and the Beth Israel Hospital held the second joint meeting of the year in the Beth Israel Hospital on November third. Dr. Louis J. Ullian presided as chairman.

The address of the evening was given by Dr. Frank H. Lahey on "The Present-Day Status of Diagnosis and Treatment of Thyroid Disease." In this presentation he did not attempt to cover the entire subject of thyroidism, but to stress some important points which are often misunderstood by the practitioner.

The first of these concerned the single, circumscribed, firm thyroid adenoma. There is a tendency among the profession to regard it as harmless. However, it is the experience of the Lahey Clinic

that ninety-six per cent. of all carcinomata of the thyroid arise in this type of adenoma. Hence, such adenomata should be removed as prophylaxis against later malignant change.

Carcinoma of the thyroid arises in about two per cent. of such cases. There are almost never local gland metastases until the capsule of the adenoma is infiltrated. However, blood-borne metastases are relatively common for there is a tendency for invasion of the blood stream in these tumors. They very often arise before there is any local metastasis.

Clinical features of the benign adenoma suggesting malignant change are a loss of the definite outline, fixity of the gland, and the development of sudden hoarseness or loss of voice.

There are several types of carcinoma. The papillary adenocystoma is remarkably sensitive to the x-ray. Even large growths may shrink after treatment to a size amenable to surgical removal. On the other hand, the small-cell, giant-cell, and adenocarcinoma are extremely resistant to x-ray.

There is another type of thyroid malignancy which has been described by the Clinic, of which about a dozen cases have been observed. This arises from lateral aberrant thyroid rests, probably from the ultimo-branchial bodies. These tumors are often bilateral, and are found, on dissection, to be anterior to the sternomastoid and great vessels of the neck. They are characterized by the presence of bluish-black, gland-like areas. They are easily dissected and, followed by x-ray, almost all are cured.

Adenomas tend to move downward on swallowing. It is by this method that intrathoracic adenomas arise. Once in the chest they tend to progress still farther since the thorax is wider below than it is at the apex. They deviate the trachea, sometimes to one side, sometimes anteriorly. For this reason, both anteroposterior and lateral x-rays should be taken of every case of intrathoracic goitre. The point of maximum diameter of the adenoma is always indicated by the point at which the trachea is most curved.

Dr. Lahey believes that it is now possible to remove all such growths without splitting the sternum. They must be freed carefully and delivered by pressure from below. Following operation the mediastinum must be drained and packed for several weeks. Fatal complications usually are due to mediastinitis.

Clinical features of the intrathoracic goitre include symptoms of direct pressure on the trachea and esophagus, and often, great dilation of the superficial epigastric, thoracic and cervical veins. This arises from direct pressure on the internal jugular veins.

The multiple colloid adenomatous goitre, the nodules of which are soft, semi-discrete and not definitely encapsulated, is the simple endemic goitre. It is seen most often in the Great Lakes region of this country. It is not a tumor, but a type of involution. Hence surgery is not indicated except for reasons other than its mere presence.

Dr. Lahey does not discriminate between toxic adenoma and exophthalmic goitre, but includes them both under the term, toxic goitre. It must be remembered that they arise in children as well as in adults, producing an exactly similar clinical syndrome. The surgeon must be extremely careful in children when resecting the gland to leave sufficient thyroid gland. Radical removal in the child is not desirable because of the danger of myxedema.

Histologically, activity of the thyroid gland is shown by absence of colloid. Whatever improves exophthalmic goitre—rest, x-ray, ligation, or iodine—produces an increase in colloid and in iodine content of the gland. Lugol's solution will improve all symptoms for six weeks to two months, and will maintain the gain for six weeks to two months more. But about six months from the beginning of the treatment, the basal metabolic rate will have risen somewhat higher than it was before treatment began. As it is extremely important that its action be conserved for preoperative treatment, physicians should not attempt to treat toxic goitre medically by this means when they are to be operated upon.

Exophthalmos needs only the slightest injury to the eye to cause an abrasion of the cornea, and loss of sight. Hence, the eyes must be carefully guarded, especially during operation. If the eye becomes edematous and infected it may be necessary to denude the lids and sew them together, or to resect the lateral wall of the orbit. Subtotal thyroidectomy should be done early in these cases to diminish exophthalmos.

"Thyrocardiacs" include those patients who show symptoms of congestive failure because of thyrotoxicosis. Such patients respond poorly or not at all to rest and digitalis, while subtotal thyroidectomy often restores compensation in a week. No patients are too desperately ill to withstand this operation. A general anesthetic such as ethylene may be used, but the patient should be sitting up during the operation.

Cardiac failure occurs as well in the myxedematous. The decompensation is frequently cleared up in these myxedematous cases by the administration of thyroxin alone.

Two groups of patients suffering from toxic goitre may be distinguished. One is the activated type—the typical exophthalmic goitre patient. Such a patient would die in activation and delirium. The other is the apathetic group. This type is the response of older age to hyperthyroidism. There are no eye signs; the hands are cool. The skin is wrinkled and pigmented, the pulse is moderately elevated, the basal metabolic rate is only moderately elevated, and the thyroid feels firm and pebbly. A patient of this type often loses a large amount of weight slowly and, if an operative fatality, dies in coma and not in activation. This group is extremely difficult and important to recognize. They

also are bad operative risks. A two-stage operation should practically always be done.

Subtotal thyroidectomy does not lower any existing hypertension. There is, however, a moderate decrease in the pulse pressure.

Some patients have low basal metabolic rates but no evidence of myxedema. They fail to respond to thyroxin. The clinic has discovered that the level of blood cholesterol will indicate those in which a response to thyroxin is to be expected. Truly myxedematous patients have hypercholesterolemia. If the cholesterol value is normal or below, thyroxin will be of no value.

With respect to the type of operation: Dr. Lahey believes that drainage is rarely necessary and should be avoided whenever possible. If done, the drain should be placed laterally running through the sternomastoid muscle. A midline scar will adhere to the trachea producing a poor cosmetic effect.

One of the undesirable results of an unskillful operation is bilateral adductor paralysis. After operation the patient can breathe easily but he cannot talk, since the cords cannot be tensed. After four months his voice returns, but he cannot breathe easily, for fibrosis has drawn the cords together. No complication of this type has arisen in the Lahey Clinic. In the patients referred for this condition, a submucous resection of the vocal cords has been quite successful in restoring their ability to breathe and to do without a tracheotomy tube.

The Clinic has recently obtained deductive proof of the presence of circulating thyroxin in the blood serum. This has been done by taking electrocardiographic records of the hearts of chicken embryos which are suspended in saline solution. If thyroxin or the serum of patients suffering from exophthalmic goitre is added to the solution, variations in the tracing are recorded, while if serum from normal individuals is added, there is no variation.

In the discussion which followed, Dr. R. W. Ohler pointed out that the differential diagnosis of hyperthyroidism and neurasthenia is extremely difficult in many cases. No relief can be obtained by operation in the latter group.

Dr. J. Lerman said that in the Massachusetts General Hospital two weeks of rest and digitalis are recommended before thyroidectomy for decompensation. Male exophthalmics are more severely affected than female, although their subjective symptoms are fewer.

In closing the discussion, Dr. Lahey stated that operations on neurasthenics are distinctly contraindicated, for it makes them worse. In the differential diagnosis careful daily basal metabolic determinations are of the greatest value. If there are great daily variations, or a steady trend toward normal, operation is probably unwise. If the case is still doubtful, the test of time shoud be employed. Hyperthyroidism should be permitted to demonstrate itself.

To save patients with thyroid storm is extremely difficult; they must be treated as soon as premonitory symptoms are observed. These include psychic changes, rise of pulse, vomiting, or diarrhea. Dehydration means crisis. Fluids, glucose, morphine, amytal or avertin and iodine must be used. The iodine in the form of Lugol's solution is of much less value.

Dr. Hurxthal has found that it is better not to employ quinidine preoperatively in auricular fibrillation due to hyperthyroidism, first, because patients with toxicity do not stand it so well as those who are non-toxic do, and secondly, because if one gives quinidine and obtains normal rhythm, the postoperative toxic reaction is very apt to bring on auricular fibrillation again. He, therefore, employs digitalis preoperatively and quinidine postoperatively after the reaction has passed.

SUFFOLK DISTRICT MEDICAL SOCIETY

The stated meeting of the Suffolk District Medical Society was held at the Massachusetts General Hospital in the Moseley Memorial Building on the evening of October 28. Dr. Reginald Fitz presided over a short business meeting after which the clinical staff of the Massachusetts General Hospital presented a symposium on infantile paralysis.

Dr. Gaylord W. Anderson, representing the Massachusetts Department of Public Health, spoke on some epidemiological aspects of the disease. The epidemic this year has been an approach to that of 1916, but it has not been nearly so severe as the newspaper reports have indicated. In fact, there have been only a few more cases this year than last. Last year the majority of the cases were in the metropolitan area but this year they have been largely in the outlying districts, especially in the Connecticut valley. During this epidemic there has been increasing evidence that infantile paralysis is often a contact infection. More secondary cases are seen in poliomyelitis than in epidemic cerebrospinal meningitis which is recognized as a contact infection. Aycock and his associates have demonstrated the existence of healthy carriers and have isolated the virus from cases and contacts. Immunity develops in certain cases analogous to that in the contact disease, diphtheria. A study of the physiological variants which spell the silent development of immunity in one case and the development of the clinical disease in another should be the next step in the attempts to understand this disease. More than 10 years ago Draper pointed out that there was a constitutional type of child predisposed to the disease but little has since been added to his observation.

Dr. Tracy B. Mallory discussed the pathologic findings outside of the central nervous system. Peabody, Douchet, and Draper originally described the usual changes found in the lymphatic system at postmortem examinations. They consist chiefly of an infiltration of the lymphoid tissues by large monocytic phagocytes. The changes are most marked in Peyer's

patches in the small intestine and in the mesenteric nodes. Burrows, in a recent paper, has suggested that this indicates that infantile paralysis is originally an enteric infection. Dr. Mallory, however, feels that these changes are the result of a generalized toxic state and do not indicate any localization of the virus.

Dr. Charles S. Kubik described the pathologic changes in the central nervous system. Grossly the pia-arachnoid is congested and thickened and may show a few small hemorrhages. Microscopically there is a degeneration of the anterior horn cells of the spinal cord accompanied by a cellular reaction with polymorphonuclear leucocytes, lymphocytes, and glial cells. There may be either a perivascular or diffuse infiltration of the cord with these elements. Often degenerated nerve cells are the center of an area of infiltration and in some cases polymorphonuclears actually invade the damaged nerve cells. Dr. Kubik considers these cellular reactions as part of a process of repair, since the invading cells often contain phagocyted particles of degenerate nerve cells. This reaction persisted for six months after the acute stages of the disease in one of the cases studied by Dr. Kubik. His remarks were illustrated by a number of lantern slides showing the gross and microscopic changes which he described.

Mr. Philip Drinker demonstrated one of the respirators which he and his associates have designed and briefly described the mechanical principles which enable it to breathe for the patient.

Dr. Neil L. Crone reported on all of twenty-three cases of infantile paralysis treated in the Drinker-Shaw respirator at the Massachusetts General Hospital. Of these, nine had intercostal paralysis, ten were of the bulbar type, and four had both types of respiratory embarrassment. Six of the first group are now alive. Three have been discharged from the hospital and of these, two have a practically complete residual quadriplegia while the third has regained a moderate degree of function in his legs. The three who remain in the hospital have been there from six weeks to fourteen months. All of these now have a complete quadriplegia and must spend a number of hours each day in the respirator. None of the bulbar and only one of the combined cases survived the acute stages of the disease. This one patient also has a complete quadriplegia at present.

Dr. S. D. Kramer briefly discussed the previous paper and introduced Mr. Hoyt, the first patient to be treated in a Drinker-Shaw respirator. After a typical prodromal stage he developed a complete quadriplegia and severe intercostal and diaphragmatic paralyses. He was almost *in extremis* when placed in the original respirator at the Peter Bent Brigham Hospital. He obtained immediate relief but was forced to spend most of the next 28 days in the respirator. His recovery was subsequently quite uneventful and he has regained almost complete function of all of his muscles. The following year he was able to return to college and graduated with honors. This case amply justifies the treatment of every case of poliomyelitis showing respiratory difficulty, in the respirator, for it is impossible to tell what the eventual outcome will be in any case. During 1930 the case fatality in Massachusetts dropped from between fourteen and fifteen per cent to approximately seven per cent. Dr. Kramer, in a personal survey, has obtained evidence strongly suggesting that nearly all of this drop in fatality can be attributed to the use of the Drinker-Shaw respirator.

Dr. H. C. Solomon discussed a group of four cases in which contact was apparently the mode of infection. Three were in one family and the fourth was an intimate friend of one of the other patients. Each showed the typical respiratory phase followed in a few days by involvement of the central nervous system. One case was of further interest as the spinal fluid was normal during the respiratory phase but subsequently showed the typical findings of infantile paralysis.

Dr. W. L. Aycock and Dr. J. B. Ayer briefly discussed the papers and following the meeting Dr. N. L. Crone and Dr. R. S. Hawkes demonstrated poliomyelitis patients in the respirators.

THE WILLIAM HARVEY SOCIETY

Wednesday, November 25, 1931, 8:00 P. M., at the Beth Israel Hospital.

Speaker: Dr. Chevalier Jackson, Director of the Chevalier Jackson Bronchoscopic Clinic, Philadelphia.

Subject: "Bronchoscopy in the Prophylaxis of Diseases of the Lungs." Illustrated with lantern slides and motion pictures.

Chairman: Dr. Harry James Inglis, Professor of Oto-laryngology, Tufts Medical School.

Dr. Jackson is regarded as a dean of bronchoscopists and has entertained the members of the Massachusetts Medical Society through his Bigelow Lecture given on October 10, 1928. The recital of his work will be a very entertaining demonstration of the "Prophylaxis of Diseases of the Lungs."

HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held in the Peter Bent Brigham Hospital Amphitheatre (Van Dyke Street entrance), Tuesday evening, November 24, at 8:15 o'clock.

PROGRAM

Presentation of Cases.

The Development of Methods for the Determination of the Numbers, Size, and Hemoglobin Content of the Red Blood Cell, by Dr. Russell Haden, of the Cleveland Clinic.

Medical students, nurses and physicians are cordially invited to attend.

JOHN HOMANS, *Secretary.*

NEW ENGLAND PEDIATRIC SOCIETY

The annual meeting of the New England Pediatric Society will be held December 11 at the Boston Medical Library at 8:00 P. M.

Dr. Homer F. Swift of New York will present a paper on Rheumatic Fever.

GERALD HOEFFEL, *Secretary.*

HAMPDEN DISTRICT MEDICAL SOCIETY

The regular Fall Meeting of the Society will be held in the rooms of the Springfield Academy of Medicine, 20 Maple Street, Springfield, Tuesday evening, November 24, 1931, at 8:30 o'clock sharp.

Papers for the evening:

"Addison's Disease," Dr. Laurence D. Chapin.

"Addison's Disease," Case Report, Dr. Ernest A. Gates.

"The Massachusetts Pneumonia Program," Dr. Roderick Heffron. Dr. Heffron is the Field Director, Pneumonia Study and Service, Massachusetts Department of Public Health.

Discussion by Fellows.

Luncheon after the meeting.

Please note that this is an evening meeting.

HERVEY L. SMITH, *Secretary and Treasurer.*

SOCIETY MEETINGS

CONGRESSES AND CONFERENCES

November 19—Medical Clinic at the Peter Bent Brigham Hospital. See page 976, issue of November 12.

November 19—New England Hospital for Women and Children. See page 983, issue of November 12.

November 19—The National Society for the Prevention of Blindness. See page 981, issue of November 12.

November 20—New England Roentgen Ray Society. See page 982, issue of November 12.

November 24—Harvard Medical Society. See page 1029.

November 25—The William Harvey Society. See page 1029.

November 30—Harvard University Medical School. Lecture on Civilization and Disease. See page 1026.

December 2—New England Obstetrical and Gynecological Society. See page 981, issue of November 12.

December 11—New England Pediatric Society. See notice above.

April 4-8, 1932—The American College of Physicians. See notice on page 1232, issue of June 4.

Autumn 1931—Spring 1932—International Medical Postgraduate Courses in Berlin. See page 468, issue of August 27.

DISTRICT MEDICAL SOCIETIES

Essex North District Medical Society

January 6, 1932—At Haverhill.

May 4, 1932—Annual meeting at Lawrence.

Essex South District Medical Society

December 2, Wednesday—Beverly Hospital. Clinic 5 P. M. Dinner 7 P. M. Speaker: Dr. Raymond S. Titus, Boston. Subject: Some Complications of Pregnancy and Puerperium.

January 6, 1932, Wednesday—Danvers State Hospital, Hathorne. Clinic 5 P. M. Dinner 7 P. M. Speaker: Dr. Harry Solomon, Boston. Subject: Modern Treatment of Syphilis.

February 3, 1932, Wednesday—Council Meeting, Boston.

February 10, 1932, Wednesday—Hawthorne Hotel, Salem. Dinner 7 P. M. Speakers: Dr. Lincoln F. Sise and Dr. Harry H. Germain. Subject: Selection of the Anesthetic for the Patient and Operation. Dr. Sise to speak from the viewpoint of the Anesthetist, and Dr. Germain from the viewpoint of the Surgeon.

March 2, 1932, Wednesday—Lynn Hospital. Clinic 5 P. M. Dinner 7 P. M. Subject to be announced later. Speaker: Dr. Elmer W. Barron, Boston.

April 6, 1932, Wednesday—Essex Sanatorium, Middleton. Clinic 5 P. M. Dinner 7 P. M. Speaker: Dr. Arthur P. Wakefield, State Department of Health. Subject: 10-Year Tuberculosis Program in the Public Schools.

May 5, 1932, Thursday—Censors' Meeting, Salem Hospital, 3:30 P. M.

May 10, 1932, Tuesday—Annual Meeting. The Tavern, Gloucester. Speaker and subject to be announced later. Ladies invited.

Franklin District Medical Society

Meetings will be held on the second Tuesday of January, March and May at the Weldon, Greenfield, at 11 A. M.

CHARLES MOLINE, Secretary.

Hampden District Medical Society

November 24—See notice elsewhere on this page.

Middlesex East District Medical Society

At a meeting of the executive committee the following schedule for the Middlesex East District Medical Society was arranged:

January—At Melrose.

March—At Reading.

May—At Woburn.

All meetings on the second Wednesday of each month unless otherwise announced.

Norfolk South District Medical Society

December 3—Speaker: George H. Bigelow, M.D., Commissioner of Public Health. Subject: Some Communicable Disease Control Problems.

January 7—Speaker: William Reid Morrison, M.D. Subject: Stomach Surgery.

February 4—This meeting will be held at the Quincy City Hospital. The program will be furnished by the members of the hospital staff.

March 3—Speaker: Roderick Heffron, M.D. Subject: Pneumonia.

April 7—To be announced later.

May 5—Annual meeting.

The time of all meetings is 12 o'clock noon. All meetings, except February 4, will be held at the Norfolk County Hospital, South Braintree. Censors will meet on November 5 and May 5 at 11:15 A. M. at the Norfolk County Hospital.

N. R. PILLSBURY, Secretary.

Plymouth District Medical Society

November 19—See page 983, issue of November 12.

Suffolk District Medical Society

December 8 - April 27—The schedule of monthly meetings of the Suffolk District Medical Society and the Boston Medical Library which will be held during 1931-32 on Wednesdays at 8:15 P. M., appears below.

December 16, 1931—Clinical Program, Peter Bent Brigham Hospital.

January 27, 1932—General Meeting in Association with the Boston Medical Library. Titles and speakers to be announced later.

February 24, 1932—Clinical Program, the Children's Hospital.

March 30, 1932—Clinical Program, New England Deaconess Hospital.

April 27, 1932—The Annual Meeting, Boston Medical Library. Election of Officers.

The Medical Profession is cordially invited to attend all of these meetings.

GEORGE S. DERBY, M.D., President.
LELAND S. McKITTRICK, M.D., Secretary.
HILBERT F. DAY, M.D., Boston Medical Library.

Worcester District Medical Society

Wednesday evening, December 9, 1931—Worcester Chamber of Commerce Rooms, Franklin Street, Worcester, Mass. 7:45—Program: Subject in the field of Eye, Ear, Nose and Throat to be announced later.

Wednesday evening, January 13, 1932—Worcester City Hospital, Worcester, Mass. 6:30—Dinner, complimentary by City Hospital. 7:45—Program: To be announced later.

Wednesday evening, February 10, 1932—Worcester State Hospital. 6:30—Dinner, complimentary by the Worcester State Hospital. 7:45—A psychiatric program to be announced later.

Wednesday evening, March 9, 1932—Memorial Hospital, Worcester, Mass. 6:30—Dinner, complimentary by Memorial Hospital. 7:45—A pediatric meeting with subjects to be announced later.

Wednesday evening, April 13, 1932—Grafton State Hospital, North Grafton, Mass. 6:30—Dinner, complimentary by Grafton State Hospital. 7:45—Program to be announced later.

Wednesday afternoon and evening, May 11, 1932—Annual Meeting. Program to be announced later.

ERWIN C. MILLER, Secretary.